1-1-1959

Volume 02, issue 2

Canadian Medical Association

(Continued from page 112)

Une technique chirurgicale qui donne 97% de consolidation dans la cure de pseudarthrose des 2 os de l'avant-bras mérite d'être suivie à la lettre; on la trouve bien décrite dans cet ouvrage.

La chirurgie du poignet fait le sujet des derniers chapitres, avec les ostéites traumatiques du membre supérieur, et enfin la rééducation fonctionnelle du membre supérieur en traumatologie.

Toute la pathologie du scaphoide carpien de même que les séquelles des fractures—luxations du poignet sont traitées avec une clarté égale à celle que l'on note aux chapitres précédents.

Les auteurs ont le grand mérite d'avoir fourni à la littérature médicale d'expression française, sur des sujets qui peuvent paraître hautement spécialisés, des notions de base indispensables à tous ceux qui traitent les traumatismes récents ou leurs séquelles. Un tel enseignement contribuera à faire diminuer les séquelles de traumatismes en nombre et en gravité, malgré l'accroissement numérique des traumatismes récents.

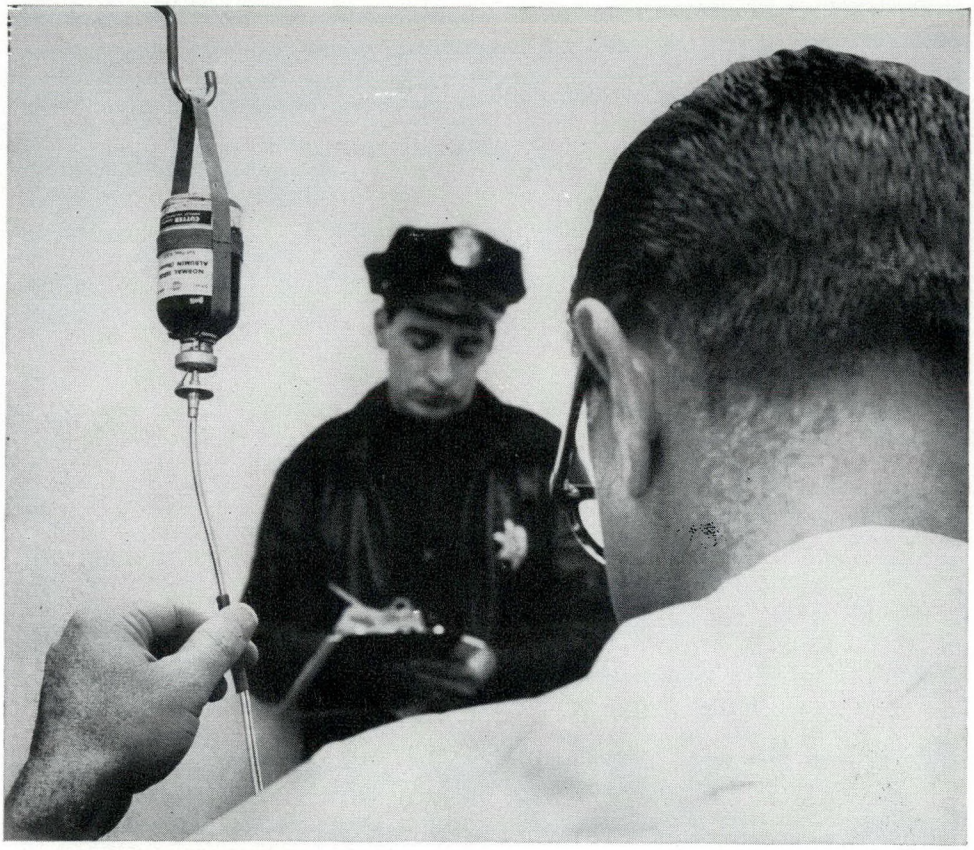
COMPARATIVE PHYSIOLOGY OF THE NERVOUS CONTROL OF MUSCULAR CONTRACTION. Cambridge Monographs in Experimental Biology, No. 8. Graham Hoyle, Lecturer in Zoology and Comparative Physiology, University of Glasgow, Scotland. 147 pp. Illust. Cambridge University Press, England; The Macmillan Company of Canada Limited, Toronto, 1957. $2.50.

Some years ago Sherrington stated: "The importance of muscular contraction to us can be stated by saying that all man can do is move things and muscular contraction is his sole means thereto." In spite of this, very little attention has been given to the mechanisms of muscular activity. The publication of Hoyle's volume as the eighth in the series of *Cambridge Monographs in Experimental Biology* is indeed welcome. This volume is a serious study of the detailed biophysics of muscular contraction. The author, however, is fully cognizant of the place these mechanisms occupy in the over-all economy of the body, and although he discusses the detailed mechanisms he does not forget that the ultimate purpose of the muscle is to contract and thus produce movement.

Much of human physiology is based on deductions from observations made on lower forms. In this volume the various observations are recorded and arranged according to species. The author sets out mechanisms developed and variations observed in the phylogenetic series. He does not extrapolate to the human, but merely presents the evidence of lower forms.

The text is well and carefully written. The bibliography is extensive but not excessive, the index small but adequate. This volume will not become a standard text for medical students but will prove a valuable asset to all who are interested in the intimate details of muscular contraction and neuromuscular mechanisms.

(Continued on page 118)



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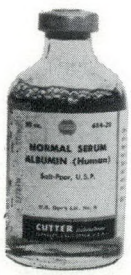


SINCE CUTTER 1897

CUTTER LABORATORIES INTERNATIONAL

106 11th Avenue, S. E., Calgary, Alberta

EARL H. MAYNARD, 207 Main Street So., Weston, Ontario

(Continued from page 116)

BRITISH SURGICAL PRACTICE: SURGICAL PROGRESS 1957. General editors, Sir Ernest Rock Carling, Consulting Surgeon, Westminster Hospital, and Sir James Patterson Ross, Director of Surgical Clinical Unit, St. Bartholomew's Hospital, London. 442 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1958. $8.00.

The 1957 progress volume is the seventh supplement to the eight volumes of "British Surgical Practice". The purpose of this is to bring recent articles, surveys and abstracts of other publications to the attention of the surgeon who wants to keep up to date. There are numerous cross references to the previous, as well as references to the original volumes of the main work. It is therefore possible by using the "Noter-up 1957" at the back of this volume, to find all the previous references to reviews and original articles that have appeared since the main work was available. Where nothing further of significance in the literature has appeared beyond what was originally published, this is clearly stated in this section.

There is a summary of important papers with their references at the end of each section; where the subject matter has been so enlarged by recent works as to make a section of the main work obsolete, it has been rewritten and included in "Surgical Progress 1957".

This little volume may therefore have a great appeal for the student preparing for examinations and alert to the most recent thoughts in surgical practice, as well as the surgeon anxious to keep abreast of the times with a minimum of time expenditure.

DUODENUM ET PANCREAS. Actualités Hépato-Gastro-Entérologiques de l'Hôtel-Dieu, 1956 (Duodenum and Pancreas. Hepato-Gastroenterological Conference of the Hotel-Dieu, 1956). Edited by Guy Albot, Hotel-Dieu, Paris, and F. Poilleux, Corentin-Celton Hospital. 296 pp. Illust. Masson et Cie, Paris, 1957. 3.500 Fr. fr.

Tout n'a pas été dit sur le fonctionnement normal du duodénum et du pancréas, mais encore moins sur le fonctionnement de ces organes après gastrectomie ou pancréatectomie. Guy Albot et Félix Poilleux nous présentent une étude poussée et très bien faite de différents problèmes qui intéressent ceux qui font de la gastro-entérologie. C'est un volume que tout chirurgien intéressé dans cette matière devrait lire.

On y lit d'abord une étude clinique et radiologique des tumeurs bénignes du duodénum, et du mega-duodénum congénital, acquis et fonctionnel. La démonstration des zones sphinctériennes de l'anneau duodénal par la double sonde duodénale et la cholécystographie, donnent des idées nouvelles sur les dyskinésies par spasme oddien.

Viennent ensuite un bel exposé des differents syndromes post-gastrectomie et une étude comparée de l'anastomose gastro-jéjunale et gastro-duodénale. Les auteurs terminent par l'étude des différents syndromes pancréatiques et leurs traitements.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Operative Surgery: Volume 7. Breast and Genito-Urinary System. Edited by Charles Rob and Rodney Smith. 273 p. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1958. $21.50.

Chirurgie d'Exérèse dans la Tuberculose Pulmonaire: Technique, Indications, Résultats (Resection Surgery in Pulmonary Tuberculosis: Technique, Indications, Results.) D. Honoré, Liège. 216 pp. Illust. Masson & Cie, Paris, 1958. 3,300 fr.

Milestones in Modern Surgery. Edited by A. Hurwitz and C. A. Degenshein, New York. 520 pp. Illust. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1958. $15.00.

Chirurgie Infantile d'Urgence (Emergency Surgery in Childhood). M. Fèvre, Paris. 718 pp. Illust. 2nd ed. Masson & Cie, Paris, 1958. 8,000 fr.

Les Déchirures Obstetricales Compliquées du Perinée, Traitement Chirurgical (The Surgical Treatment of Complicated Obstetrical Perineal Ruptures). R. Musset et al. 120 pp. Illust. Masson & Cie, Paris, 1958. 1,400 fr.

Human Blood in New York City: A Study of its Procurement, Distribution and Utilization. Conducted by the Committee on Public Health. The New York Academy of Medicine, New York, 1958.

Clinical Obstetrics and Gynecology: Vol. 1, No. 2. Fibromyomas of the Uterus. Edited by R. A. Kimbrough. Toxemias of Pregnancy. Edited by L. M. Hellman. 249 pp. Illust. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1958. $18.00 per vol.

An Introduction to Experimental Surgical Studies. W. J. Dempster. 463 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1957. $12.00.

Extracorporeal Circulation. Compiled and edited by J. Garrot Allen. 518 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1958. $8.25.

Anomalies of Intestinal Rotation and Fixation. R. L. Estrada. 161 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1958. $7.25.

Lehrbuch der Chirurgie (Textbook of Surgery). M. Allgoewer. Edited by H. Hellner and others. 2nd ed. 1112 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1958. $20.00.

Allgemeine und Spezielle Chirurgische Operationslehre: Erste Band, Teil I (General and Special Surgical Procedures: Volume I, Part I). Gerd Hegemann. Edited by N. Guleke and R. Zenker. 420 pp. Illust. 2nd ed. Springer Verlag, Berlin, Göttingen, Heidelberg, W. Germany, 1958. DM 496 (Parts I and II).

HISTORY OF CANADIAN SURGERY

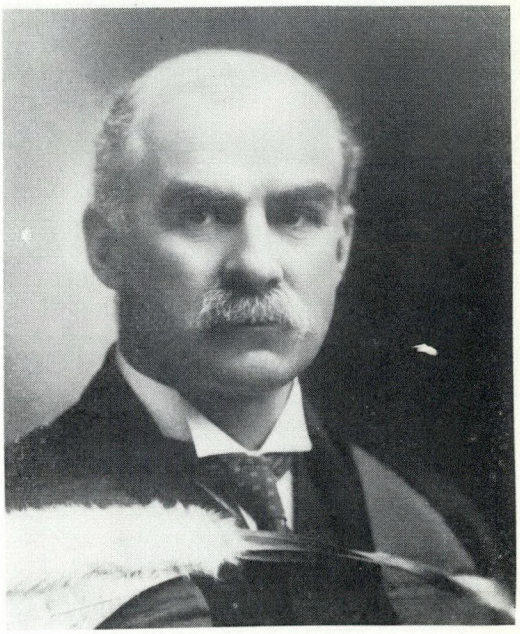
GEORGE ARMSTRONG PETERS
(As I Remember Him)

W. E. GALLIE, M.D., *Toronto*

THIS INVITATION to write a note on Professor Peters is a very acceptable one, for it not only enables the writer to recall the delightful experience of his association with him, but it makes it possible to link together two periods of the history of our Toronto school and hospitals which are separated by over 50 years.

In attempting to paint a picture of old Geordie Peters, as I remember him, I must try to forget the intervening half century, and transport myself back to the days when I was young and overwhelmed by the personality of this remarkable man. I was one of his last house surgeons, both at the Hospital for Sick Children and at the General Hospital, and the influence he had on me during those two years decided the type of career that I would follow. He was the best surgeon and surgical teacher that I ever knew, quite outclassing those of Toronto and New York and Great Britain with whom I came in contact. This impression is just as strong today as it was half a century ago.

One of the first things he taught us was how to sew up wounds. Graduating as he did in 1886, it is surprising that he showed no enthusiasm for antiseptics. His contacts with Lister must have come when the carbolic acid spray had been abandoned and the principle of asepsis adopted. At any rate, Peters used a technique very similar to what we have today. Because of Lister's experiments, he took every precaution to avoid bruising and tearing the tissues, to prevent the collection of blood and serum in the closed wound, and to eliminate the possibility of infection from damaged skin. Peters' subcuticular horsehair suture, when carefully inserted, left a wound that would have pleased the modern plastic surgeon. The healing of his wounds was the pride of the hospital and has not been improved upon since.



Dr. George Armstrong Peters.

As a clinical teacher he was superb. One day he called me over to a child with a distended abdomen and asked me to put my stethoscope on the lower half of the abdomen and tell him what I heard. I listened for a while, expecting to hear intestinal gurgling, but before long became aware of the loud beating of the heart. Then he suggested that I listen for breath sounds and sure enough they were quite audible. This was "Peters' sign" for general peritonitis.

This incident illustrates his method of clinical teaching. He rarely told us anything but rather led us to make the observation ourselves. By the time I got through with that patient, I knew more about peritonitis than a library of books or a series of lectures could have taught me.

The way habits learned in youth stick to one through life is amazing. One day during an appendectomy my "resident" asked me how long I had been turning back a

sleeve at the base of the appendix before ligating and severing it. My answer was that I had learned it from George Peters, nearly 40 years before, and that I didn't know there was any other way of doing it.

While I was with Peters at the Hospital for Sick Children he was intensely interested in devising a safe treatment for that dreadful malformation of the bladder, ectopia vesicæ. His operation consisted of the transplantation of the ureters into the rectum extraperitoneally, thus avoiding the great risk of peritonitis which attended other operations such as Madyl's in which the ureters are transplanted into the colon intraperitoneally. This Peters operation is an excellent one, in that it is easy to do and it eliminates completely the risk of peritonitis. What the long term results may be is uncertain, for the risk of ascending infection sooner or later must be great, but I do know that I saw some of these patients several years after their operations, completely changed from a condition of abject misery to one of comparatively happy childhood. They had gradually developed control of the rectum so that the urine could be retained for several hours, without mishap.

Peters was, above all things, a general surgeon. One could not imagine him allowing himself to be cornered into some specialty. The whole field of anatomy was so interesting to him that he could not be content with a part of it.

Following an internship at the General Hospital he joined the staff of the Department of Anatomy and quickly became one of its most brilliant teachers. In this, his skill with chalk and pencil was of great value. I have before me now a collection of his pocket case-books which are full of sketches and water colours which vitalize the written word.

After a spell in the Anatomical Department he went to England and immediately passed the primary examination for the Fellowship. Six months later he tried and passed the final. He was one of the first Canadians to become a Fellow of the Royal College of Surgeons and he did it with a flourish.

Upon his return from England he was promptly appointed to the Department of Surgery and to the staffs of the General and the Children's Hospitals. These appointments marked the beginning of a brilliant surgical career, illuminated from time to time by studies of unusual cases and by reports of the surgical treatment of such pathological conditions as hydatid cyst of the tail of the pancreas, fusiform dilatation of the œsophagus, œsophageal foreign body, procidentia recti, ascites due to cirrhosis of the liver, ectopia vesicæ, and others.

Quite aside from his skill in the diagnosis and treatment of disease, he had a remarkable gift for designing and perfecting mechanical devices to be used in surgery and elsewhere. Of these his wrench for the correction of stubborn deformities, and his method of cutting urinary calculi, are well known. But the one that interested me most was described in his paper on "A New and Original Method of Making Casts" (*Brit. M. J.*, Sept. 3, 1898). This was a device whereby he was able to spray an object with liquefied paraffin wax which, when thickened and cooled and supported by a coating of plaster-of-Paris, could be removed in sections and subsequently fitted together again to form a mould. This mould was then filled with liquid plaster-of-Paris and allowed to solidify. It is now that the value of the method becomes apparent, for in place of the former difficult and unsatisfactory method of chipping off the mould with hammer and chisel, one simply immerses the whole mould and cast in hot water. Promptly the wax mould is melted and the cast becomes quite free.

The method as presented originally to the Surgical Section of the British Medical Association in Edinburgh in July 1898, had the disadvantage of requiring expensive apparatus. But for the making of casts that do not require the absolute perfection of show pieces, he obtained satisfactory results by applying the liquid wax with a soft brush and covering it with plaster-of-Paris which could be divided into sections by cutting out silk threads which had been laid under the wax. This method has stood us in good stead for the casting of feet along with the object on which the patient was standing.

Peters was a man who believed that a surgeon should have some other interest

aside from his work. His had to do with horses. He always kept a stable of excellent drivers and hunters, and my friends told me that there was no more daring and skilful cross-country rider at the Hunt Club than George Peters. I can remember him well, driving snappily up to the old General Hospital on Gerrard Street with his groom sitting up behind him.

This interest in horses led to his joining the Governor-General's Bodyguard and subsequently to his organizing the Toronto Light Horse, of which he was Colonel.

His interest in the army led to his inventing the Peters self-registering electric target. This was a most ingenious idea which provided that the target 200, 400 or 600 yards away was connected by electrical wires with a miniature target at the right hand side of the marksman. When the shot hit the distant target it also registered on the miniature target close at hand.

Peters' gift for invention was very materially aided by his close friendship with Philip Rensman, the chief mechanic in the machine-shop at the Children's Hospital. Rensman was a superb mechanic, who was not only able to produce equipment but was also able to make valuable suggestions towards making the ideas practical.

In reading over his papers, I have been struck once more with the excellence of his literary style. This is apparent not only in the short papers presented to the medical societies but also in the longer contributions he made to Bryant and Buck's *System of Surgery* on the "Inflammatory Affections of Bone", and the "Surgery of the Rectum and Anus" in the *International Textbook of Surgery*. He made no effort to be decorative in his style but he made perfectly sure that his audience and readers understood everything he said.

He died on March 13, 1907, at the age of 47 years, of angina pectoris. Professor Cameron has recorded that even when he knew the end was approaching he dictated to his stenographer a description of the radiating pains of this dread disease as exemplified in his own case, and pointed out where they differed from the ordinarily accepted ideas. That is the kind of man he was.

He was buried with full military honours, but the sight of his horse without its rider and with his boots in the stirrups, reversed, blotted out for me all recollection of that great funeral. I have been listening to the "Last Post" ever since.

But 50 years have rolled around since that last sad day, and it is now my happy privilege to hand on to my young colleagues an impression of one of the great founders and builders of our school. As I said before, he was the best surgeon and the best surgical teacher I have ever known. It may be thought that this is the exaggerated estimate formed by a worshipping youth, but I assure you that it was also the opinion of such men as Irving Cameron, Alexander Primrose, Clarence Starr, Fred Starr and George Bingham, who were more or less his contemporaries. He was more, indeed, than a great surgeon and a great teacher for he was also a great man. Reading over the obituary notices published in 1907 and several of the tributes paid to him at that time, one is inclined to smile at the emphasis placed on gentleness and sweetness of disposition. Certainly he was courageous, honourable, and kindly, but he was far from being gentle and sweet dispositioned. A glance at his photograph, which by the way is an excellent one, indicates at once the kind of man he was. In the operating-room, as on the parade ground, any incompetence or slothfulness on the part of his assistants would bring a roar from him that would shake the windows. At any rate his house surgeon's knees shook. Fortunately his intolerance of incompetence was the kind that stimulated in his students and assistants an intense desire to do the job well.

Peters served on the Department of Surgery from 1892 to 1907. From 1903 to 1907 he was Professor of Surgery. His memory is kept alive among the students by the George Armstrong Peters Prize, which was established in 1912 by his friends. This prize which consists of a sum of money and an engraved piece of sterling silver is awarded to that graduate of the University of Toronto, of not more than 15 years' standing, who has made a sufficiently important contribution to surgical science. The list

of the winners includes the names of many of our most brilliant graduates.

Fifty years is a long time and one's memories are inclined to grow dim. But this look back into the past has been a happy occasion for the writer, for it has given him an opportunity to record something of importance in the history of our school and to give a final salute to one to whom we all owe so much.

607 Medical Arts Bldg.,
170 St. George Street.

LES DEBUTS DE L'ASEPSIE

Dans un numéro de *Médécine de France* (No. 96, 1958) le professeur Antonin Gosset rappelle que la technique de l'asepsie a été conçue dans les vieux bâtiments historiques de la Salpêtrière à Paris par un maître trop oublié: Félix Terrier.

"En arrivant à Bichat en 1882, Félix Terrier n'était pas satisfait de ce qu'il avait réalisé à la Salpêtrière, il sentait qu'il n'avait pas encore de 'méthode', et c'est alors qu'il prit la résolution si fructueuse pour la chirurgie française d'aller s'initier dans le laboratoire Pasteur, rue d'Ulm. Il m'a bien souvent raconté ses visites chez Pasteur et a bien souvent manifesté devant moi sa reconnaissance envers le docteur Roux auquel Pasteur l'avait confié. A mon sens, c'est à ce moment que se place l'événement capital de la vie de Félix Terrier.

"C'est en 1882 qu'il a fixé tout son programme: transporter l'esprit bactériologique dans les salles d'opération, c'est à dire travailler dans une salle d'opération comme on le fait dans un laboratoire de bactériologie, et pour cela n'utiliser que des objets stérilisés; mais stériliser un objet ne veut pas dire l'avoir simplement fait bouiller, car l'ébullition n'est pas un moyen suffisant, ce n'est pas un moyen mathématique. Et Félix Terrier, avec son esprit absolu, voulait que la stérilisation fût non pas un calcul de probabilité, mais une certitude.

"En 1887, un de ses élèves, Poupinel, invente une sorte de four à flamber qui peut être employé dans les salles de stérilisation, est facilement utilisable et marche à la simple chaleur du gaz d'éclairage. En 1889, à propos de la publication de sa statistique, Félix Terrier revient sur les avantages du 'Poupinel' qu'il emploie dans son service depuis le début de 1888. Cet appareil, qui a été employé peu à peu dans tous les grands services de chirurgie en France, depuis 1887, et qui est même encore employé dans certains services aujourd'hui, avec la modification heureuse que mon collègue et ami, le docteur Wiart, chirurgien des hôpitaux, lui a fait subir en le chauffant électriquement, cet appareil de Poupinel ne fut pas accueilli par tous favorablement à ses débuts.

"A la communication de Félix Terrier disant qu'il a adopté la stérilisation à l'air sec au moyen du Poupinel, parce qu'on peut la porter, au moins pour les instruments, au-dessus de 150 degrés centigrades, un des collègues de la Société de Chirurgie lui répond: 'Je ne prétends point critiquer le procédé que M. Terrier préconise, mais celui des ébullitions successives dont je me sers (ébullition de dix minutes répétée deux fois à trois ou quatre jours d'intervalle) est peut-être plus pratique que tout en conduisant au même but (O. Terrillon).'

" 'Le reproche que j'adresserai au système de M. Terrier, c'est qu'il nécessite une installation et un outillage tout spéciaux (O. Terrillon).'

"Vous voyez les objections apportées à une chose nouvelle qui réalisait vraiment un énorme progrès, permettait d'atteindre sans inconvénient des températures qui n'avaient jamais été atteintes auparavant! Vous voyez, comparée à la simplicité de manœuvre du Poupinel, l'ébullition de dix minutes, répétée deux fois à trois ou quatre jours d'intervalle!

"Vous voyez aussi le reproche perpétuel que nous retrouvons toujours en chirurgie, formulé contre tout progrès: une installation et un outillage tout spéciaux! Mais sans installation et outillage tout spéciaux, que pourrions-nous faire!"

ORIGINAL ARTICLES

HIRSCHSPRUNG'S DISEASE NINE YEARS' EXPERIENCE AT THE HOSPITAL FOR SICK CHILDREN, TORONTO

BERNARD LANGER, M.D.,* and STUART THOMSON, M.D.,† *Toronto*

Development of the Modern Concept of Etiology

Hirschsprung's disease, or congenital megacolon, has been recognized as a specific disease entity for well over 70 years. However, it is only in the past 15 years that a satisfactory concept of its etiology and treatment has evolved.

In 1888, Hirschsprung,[12] a Danish pædiatrician, reported two cases of chronic constipation and intermittent obstipation in children which went on to complete obstruction and death at the ages of seven and 11 months respectively. The post mortem findings were tremendous dilatation and hypertrophy of the colon down to the rectum without obvious mechanical obstructive factors. By 1904, he had collected another eight cases and a search of the literature disclosed 55 additional cases which fitted the clinical picture. On the basis of his observations in the post mortem room, Hirschsprung felt that this disease was a primary congenital dilatation and hypertrophy of colon, or a congenital giant colon.

After Hirschsprung's report, other clinicians began reporting cases, and additional theories regarding etiology arose. Marfan,[13] in 1895, noted that there was increased length and redundancy of the sigmoid in these cases, and thought that the dilatation was due to chronic constipation secondary to kinking of the sigmoid colon, causing mechanical obstruction. The third theory was that of defective innervation of colon, and there were at least two variations on this theme. Fenwick[8] believed that the basis of the disease was anal spasm, that is, increased tone distal to the dilated colon. Lennauder held that dilatation resulted from defective innervation to the dilated segment, causing decreased tone proximally. He went so far as to give one patient "electric enemas" in which he attempted to stimulate the sigmoid with a faradic current.

Thus except for the few who clung to anal spasm as a cause, physicians in general considered the dilated segment to be the site of the disease on the basis of one of the following: congenital dilatation, mechanical kinking and obstruction, or defective innervation.

The observation of anatomical changes in the distal undilated segment was made early in the history of the disease, but the significance of these changes was unfortunately not appreciated. Dalla Valle[5] in 1920 was the first to note changes in nerve cells in the myenteric plexus of the rectum in a case of Hirschsprung's disease. In 1928, Cameron[4] described similar changes in several more cases. These observers noted sparseness of ganglion cells of Auerbach's plexus and also described degenerative changes in those ganglion cells present. They felt, however, that these were secondary phenomena caused by chronic constipation with resulting inflammation producing degeneration.

Robertson and Kernohan[22] in 1938, and Tiffin, Chandler and Faber[23] in 1940, again described these same pathological findings and suggested that Hirschsprung's disease was due to a physiological abnormality of motility related to the absence of ganglion cells. These ideas were not generally accepted, and as recently as 1946, Ehrenpreis,[6] in his classical monograph, described Hirschsprung's disease as a "dysfunction of evacuation of the colon, of as yet unknown origin, occurring in the absence of morphological and mechanical causations, and giving rise secondarily to a characteristic dilation of the colon".

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*Resident Surgeon, The Hospital for Sick Children,
†Associate Surgeon, The Hospital for Sick Children, Assistant Professor of Surgery, University of Toronto.

It was not until 1948 that Swenson[15-17] correlated the pathological findings with x-ray observations, and in addition carried out motility studies on the colon and presented the hypothesis which comprises our present concept of the disease.

Swenson stated that the primary defect is a congenital maldevelopment of the innervation to the distal undilated segment of bowel. This segment is recognizable on pathological examination by absence or scarcity of ganglion cells in the myenteric plexus, and usually accompanied by an increase in the number of nonmyelinated nerve fibres. He stated that this defect in innervation was responsible for the failure of this part of the bowel to expand or transmit peristaltic waves in a normal fashion, thus producing a physiological bowel obstruction with resulting proximal dilatation and hypertrophy. The aganglionic segment in most cases involves the rectum and rectosigmoid region, but in a significant number of cases, involves a large part or all of the colon and may extend into small bowel.

The exact nature of this autonomic nervous malformation is not known, since the normal mode of formation of myenteric plexus in the embryo is unknown. The most promising, though not completely satisfactory, hypothesis is that of Bodian[1, 2] who postulates that neurons of the myenteric plexus form in a cranio-caudal direction, as outgrowths of the central nervous system, and that arrest of this process at some stage results in an aganglionic bowel distally. Swenson, on the other hand, believes the primary defect to be a failure of development of the pelvic parasympathetic outflow with secondary absence of neurons in the bowel wall.

Incidence

The relative incidence of Hirschsprung's disease is estimated by Bodian to be 1 in 20,000 to 30,000 live births, making it about one-quarter as common as fibrocystic disease.

All series show marked male predominance, the proportion being somewhere between 80% and 90% males.

A familial tendency was suggested by some early reports including that of Zuelzer and Wilson,[24] who described five cases in one family of 12 siblings. Bodian, Carter and Ward[2] in a careful follow-up of 40 cases found the disease in four siblings including one set of twins, and estimate that where a case of Hirschsprung's disease occurs in a family, the chance of a subsequent male child being affected is about one in five.

Clinical Features and Diagnosis

The clinical picture of the disease may be considered under two headings:

1. The classical form, which has been described and well known since the time of Hirschsprung.

2. Neonatal megacolon, which has been recognized only in the last 10 to 15 years.

1. *Classical Hirschsprung's disease* presents in the vast majority of cases with a history dating from the time of birth. The child may be anywhere from one month of age upward when first seen but is usually less than one year old. The story is that of chronic constipation and abdominal distension of varying degrees. The patient may present with chronic constipation alone, with constipation and intermittent bouts of subacute obstruction, or with a history of progressive constipation and distension going on to complete bowel obstruction. These children have a greatly distended abdomen through which fæcal masses can be palpated and often seen. They are often poorly nourished, and physically underdeveloped. The rectum is usually, though not always, empty of fæces.

The plain x-ray film demonstrates dilated loops of bowel containing fluid fæcal material and gas. The barium enema examination, when properly carried out by the technique of Neuhauser,[16] demonstrates the narrow aperistaltic, aganglionic segment and proximal dilated bowel.

2. *Hirschsprung's disease in the newborn* was first described as an entity by Ehrenpreis[6] in 1946, who reported 10 cases followed up from birth with x-ray findings. Since then, there has been much interest in the diagnosis in infancy, and numerous case reports have been published. As a result, cases are now picked up shortly after birth which might otherwise have gone unnoticed until much later.

The presenting symptoms in the first few days of life are usually delay in the passage of meconium, with early vomiting and varying degrees of abdominal distension. This early episode of obstruction may be relieved by spontaneous passage of stool, or by a movement after an enema or even after rectal examination. The child may then appear well, only to present at a later date with the classical features of megacolon. On the other hand, the newborn with Hirschsprung's disease may continue to have intermittent partial obstruction going on to complete obstruction and death in the early days of life if untreated.

The barium enema does not as a rule reveal the characteristic dilatation, since this usually takes weeks or even months to develop. Significant points to be observed, however, in the barium enema examination in the newborn period are the poor distensibility of the lower bowel during the filling phase of the enema and also the impaired ability of the bowel to evacuate the barium. Evans and Willis[7] recommend the taking of 24 and 48 hour films. The finding of large amounts of barium still in the bowel after 24 hours is strongly suggestive of Hirschsprung's disease in the absence of any mechanical obstruction.

An important symptom which may occur in either the neonatal or in the older age group is diarrhœa. This is sometimes a spurious diarrhœa with leakage of small amounts of stool around a fæcal impaction, but is more often enterocolitis secondary to chronic impaction plus inflammation and mucosal ulceration.

The final diagnosis is always made by pathological examination demonstrating the absence of ganglion cells in the myenteric plexus. Extra-mucosal biopsies may be taken when the abdomen is open either at the time of the preliminary colostomy, or when the pull-through procedure is being done. Swenson[18] has described a method of biopsy of the muscular wall of the rectum to make a certain preoperative diagnosis in cases which are not typical clinically and radiologically. Bodian, on the other hand, biopsies the rectal mucosa and relies for a diagnosis on the observation of changes in the submucosal plexus similar to those seen in the myenteric plexus. The difficulty in the former technique is in getting an adequate specimen of muscle for examination. In carrying out the latter technique, there is no trouble in getting the mucosal biopsy; the difficulty lies in its interpretation. Because of these difficulties, Hiatt[11] has recently been using a posterior approach to the rectum through a small transverse incision midway between anus and coccyx, in an attempt to get a more satisfactory specimen of rectal muscle wall without entering the lumen.

Treatment

The treatment of Hirschsprung's disease was notably unsatisfactory before the development of the present concept of the disease.

1. *Medical treatment* with diet, enemas and parasympatheticomimetic drugs such as methacholine hydrochloride (Mecholyl) was of some help in the very mild cases.

2. *Surgical treatment* consisted in either: (a) a left lumbar sympathectomy, which was done in an attempt to increase the tone of the proximal segment, or (b) surgical excision of part or all of the proximal dilated colon, in the belief that this dilated bowel was the primary site of the disease. Some successes were reported with these surgical procedures, but the results were usually only temporary.

With the recognition of the distal aganglionic segment as the diseased part of the bowel, Swenson also developed a method of excising the distal segment as far as the anus and anastomosing ganglion-containing bowel to anus by a pull-through procedure.[15] This operation has now replaced all other procedures in the treatment of the disease. In his early cases, Swenson performed the pull-through operation as a primary procedure except where the condition of the child would not permit, in which case he made a preliminary defunctioning colostomy. Because of the difficulty in operating on tiny infants, he now recommends preliminary colostomy when the diagnosis is made in a child under 12 to 18 months of age, with delay of definitive surgery until it reaches that age. In older children, he performs a primary pull-through operation if the child's condition permits.

There has been some concern about damage to the pelvic parasympathetic nerves during the operation with resulting impotence in males. Swenson has recently reported a follow-up study on 200 pull-through procedures, including 14 in males who have since married.[21] Eight of these have had children, and the other six deny impotence.

Review of Cases at The Hospital for Sick Children, 1949-1958

A total number of 58 cases were seen. The diagnosis in most was verified by pathological examination; the rest were diagnosed on clinical and radiological grounds with pathological material not yet obtained. There was a marked male predominance as in other reported series. Forty-six of the cases seen were in males, 12 in females.

TABLE I.—Age at First Hospital Admission

1 - 7 days	20 cases
8 days - 1 month	10 "
1 month - 1 year	18 "
Over 1 year	10 "
	58 cases

The age at which symptoms brought these children to the hospital for the first time varied from one day to six years. One-third of all cases presented in the first week of life, and one-half were seen by the end of the first month. A total of 83% of the patients had been admitted to hospital at least once by the time they were one year of age (Table I).

The problem of diagnosis in the newborn is reflected in the fact that of the 20 cases seen during the first week of life, the correct diagnosis was made within a week of admission in only five . Of the remaining 15, five were recognized at post mortem and 10 were diagnosed after a period of time varying from two weeks to six and a half years. The provisional diagnoses made in these 15 cases are listed in Table II. Two of the children thought to have had rectal bands were presumed cured on their first admission when spontaneous evacuation and relief of distension followed digital rectal examination.

TABLE II.—Cases of Hirschsprung's Disease in the Newborn
Diagnosis Delayed or Missed

Provisional diagnosis	*No. of cases*
Bowel obstruction (unspecified but not including Hirschsprung's disease)	5
Meconium ileus	3
Congenital anal stenosis or rectal band	5
Gastroenteritis	1
Pyloric stenosis	1

Mortality

There were 20 deaths in this series of 58 patients—an overall mortality of 37%. An analysis of these deaths (Table IV) shows, however, that only eight are postoperative deaths, and four of these eight patients were moribund at the time of operation. Twelve of the 13 patients who had no suitable operation died. There was no difference between the overall mortality in the neonatal cases and in those seen after the first week of life.

The level of the aganglionic segment was estimated by the pathologist in most cases and by x-ray where a pathological specimen was not available. It should be noted, however, that the x-ray level does not always correspond to the pathological level. Frequently the upper end of the aganglionic segment becomes somewhat dilated. Just proximal to the totally aganglionic area is a transitional segment with scanty ganglion cells which is almost always dilated.

TABLE III.
Extent of the Aganglionic Segment

	No. of cases	*Deaths*
Rectum to rectosigmoid	34	11
Sigmoid colon distally	13	2
Splenic flexure distally	6	2
Entire colon	1	1
Colon and terminal ileum	3	3
Colon, ileum and most of jejunum	1	1

Of the 58 cases reviewed, the aganglionic segment in 47 (or 80%) was rectum alone, or rectum and sigmoid. These comprised the short segment cases. The remaining 11 long segment cases involved bowel proximal to sigmoid colon (Table III).

It is interesting to note that cases with long segment involvement did not necessarily present with early severe symptoms. Those with segment involvement up to the splenic flexure were first seen in hospital

anywhere from two days of age to six years, and of the five cases involving the entire colon with or without part of small bowel, two were not seen until two and three months of age respectively. This suggests that there are different degrees of severity of the functional obstruction produced by the disease, quite independent of the length of bowel involved. Thus one patient with a rectosigmoid segment involvement may develop complete obstruction requiring emergency operation in the first week of life, whereas another with identical involvement may carry on for many years with laxatives and enemas alone.

The overall mortality in those cases with an aganglionic segment extending up only to the splenic flexure was comparable to the overall mortality in the short segment group. All five patients who had the entire colon with or without small bowel involved died.

Four of these last five cases have been reported by Bowden, Goodfellow and Munn, of The Hospital for Sick Children.[3] In their paper they point out the difficulty involved, first in making the diagnosis at all in the long segment case, and then, in differentiating the long from the short segment cases in infancy, where the characteristic dilatation has not yet occurred. In those infants in whom the entire colon is involved and the diagnosis is made, they stress the importance of an immediate ileostomy with the later choice of either a permanent ileostomy, or colectomy and ileo-anal anastomosis by the pull-through technique. Sandegaard[14] has reported one such case successfully treated by this operation, and Swenson[20] has two.

Diarrhœa as a presenting symptom was noted in eight cases in this series. The incidence was a little higher in the long segment group—three of 11 cases (27%), as compared with five of 47 cases (10%) in the short segment group. Four of these eight children died without operation, two of bowel obstruction and two of enterocolitis.

Urinary abnormalities have been reported by Swenson[19] as occurring with significant frequency in association with Hirschsprung's disease. Swenson has intravenous pyelography, cystography and cystometrography performed on all cases. He reports that 50% of cases show increased bladder capacity and decreased detrusor activity in the cystometrogram, and 5% have dilatation of the upper urinary tract, or megaloureter, demonstrable on intravenous pyelogram. He feels that the identical pathological process present in the bowel accounts for these urinary abnormalities, namely, defective innervation with dysfunction of the lower urinary tract associated with a decrease in ganglion cells in the wall of the bladder and the lower ureters.

In this series of 58 cases, intravenous pyelography was done in 14. Thirteen of these pyelograms were interpreted as normal. One case had dilatation of bladder and ureter preoperatively which returned to a normal pattern postoperatively. This was felt to be a case of chronic lower tract obstruction secondary to external pressure from a dilated colon.

Treatment

Of the 58 cases seen at The Hospital for Sick Children in the past nine years, 45 were treated operatively by one of the following: defunctioning colostomy alone; Mikulicz's resection; pull-through procedure.

TABLE IV.—Method of Treatment and Mortality

	No. of cases	*Deaths*
Defunctioning colostomy only	15	5
Mikulicz resection	11*	1
Pull-through procedure	22	2
Miscellaneous (non-operative)	13	12
	61	20

*Three of these subsequently underwent pull-through procedures.

The remaining miscellaneous group consisted of those cases which received no treatment, medical treatment, or inappropriate surgical treatment. These are considered as the non-operative group (Table IV). The diagnosis in five of these was not made until post mortem. There were 12 deaths in this group of 13 cases. Nine died of bowel obstruction (in two of these abdominal exploration was negative). Two died of enterocolitis complicating subacute bowel obstruction, and one died of perforation after excision of what was thought

to be a stenotic terminal ileum. This last case proved to be one of long segment Hirschsprung's disease with the entire colon and terminal ileum aganglionic.

Of the 15 patients treated by defunctioning colostomy alone, three were moribund on admission and subsequently died. One patient had repeated evisceration and died. One patient died after postoperative perforation, the colostomy having been made in the aganglionic segment. Ten children are alive and well with a colostomy and are awaiting admission for their pull-through procedure when they reach the required weight.

Eleven Mikulicz resections were done in this nine year period. Three cases had no recurrence of symptoms in a limited follow-up. In five cases, symptoms recurred postoperatively. Two of these are being managed medically, and three have required subsequent pull-through procedures. There was no follow-up in two cases, and one child who suffered a perforated colon after an enema had an emergency Mikulicz resection but died shortly after. Mikulicz resection is not considered a satisfactory operation for Hirschsprung's disease and is no longer done in this hospital.

The first pull-through procedure in this hospital was done in 1949, and since then a total of 22 patients with Hirschsprung's disease proven by pathological examination have undergone operation. The age at time of operation varied from one month to 11 years. In the early 1950's the operation was a primary procedure, without preliminary colostomy, at any age from one month on, if the patient's condition permitted. During recent years, the practice has been to perform a primary pull-through operation only in the older group of children (over two years of age) who are in good condition. In all others, a preliminary defunctioning colostomy is done above the aganglionic segment, and in infants, a colostomy is done and the definite operation is delayed until they reach at least 20-25 lb. in weight.

Of the 22 cases (Table V), six have been followed up for less than six months, which is not considered a long enough period for assessment. Of the remaining 16 cases, 11 are considered good results. These have

TABLE V.—Pull-Through Operations in Proven Hirschsprung's Disease

Results of operation—22 cases done	
2 postoperative deaths	
14 cases followed-up more than six months	11 good results
	3 fair results
Postoperative complications:	
Leak at the anastomosis	6
Stenosis at the anastomosis	4
Postoperative eventration	2
Bowel obstruction—adhesions	2
Recurrence—inadequate resection	1
Peritonitis—operative contamination	1

spontaneous movements without need for a routine of laxatives or enemas, and without fæcal incontinence. These have been followed up for six months to nine years. In three cases results are fair. These require either dilatation for stenosis, or occasional laxatives and enemas to maintain regular movements, but have had no chronic distension or obstruction.

There were two postoperative deaths after the early operations done in 1950. One followed gross contamination of peritoneum at operation; the other followed a postoperative leakage with peritonitis. These children were one month and four months old respectively and neither had a defunctioning colostomy.

The complications of the operation are outlined in Table V. Of the six patients with a leak at the anastomosis, four had spontaneous closure, one had a complete separation of the anastomosis and required secondary operative repair, and one developed peritonitis and died. Postoperative anastomotic stenosis has been relieved by a short period of dilatations in two cases, but in another three has persisted despite dilatations for four months, two and four years respectively. The cases of postoperative eventration or postoperative obstruction due to bands have all done well. One child had a recurrence of his symptoms due to inadequate resection of the distal end of the aganglionic segment. He had a repeat pull-through operation and the result is now good. The peritoneal cavity was contaminated in one infant and this accounts for the second postoperative death.

In reviewing these 58 cases of Hirschsprung's disease, another six cases were encountered in which a clinical diagnosis of megacolon had been made and the pull-through operation carried out, only to have

the surgical specimen reported as containing ganglion cells from end to end. These have not been included as part of the series on the basis of negative pathological findings. The history was suggestive of Hirschsprung's disease in all these cases. The barium enema was thought to demonstrate a "narrow segment" in four cases. The other two were operated upon on the basis of a positive report on a rectal mucosal biopsy in one, and the gross appearance of a narrow segment of bowel at laparotomy in the other. The results in all were good. It is difficult to be sure whether these children had functional megacolon or true Hirschsprung's disease, with a very short segment, which was left in.

The technique of the pull-through operation as practised now includes the taking of extramucosal biopsies at the time of operation, in order to confirm the clinical diagnosis, and to select a point of resection proximal to the aganglionic segment. This technique, however, does not produce a sample of the portion of rectum below the pelvic floor, and if this is the only part of bowel without ganglion cells, the diagnosis cannot be made by these means before carrying out the pull-through operation. Rectal biopsy is a method of sampling this most distal part of bowel, which is always aganglionic in Hirschsprung's disease. This procedure is not necessary where the diagnosis has been verified by pathological examination at the time of preliminary colostomy, but it may be invaluable in those children who have had a colostomy in infancy without a pathological diagnosis and who return in a year or two for definitive surgery.

Rectal biopsy by Swenson's technique has only recently been attempted at this hospital. Eight biopsies have been done in the past one and a half years. A satisfactory specimen was obtained in only four cases. In the other four cases the specimen was inadequate and the pathological report accordingly unhelpful and in one case misleading.*

*Since this paper was originally prepared, two more cases have been encountered in which a satisfactory rectal biopsy was not obtained using Swenson's technique. A second attempt using the posterior approach as described by Hiatt[11] was successful in both cases.

Summary

The development of the present concept of etiology and treatment of Hirschsprung's disease is reviewed.

Fifty-eight cases seen in this hospital in the past nine years have been studied.

With a better understanding of the disease, the diagnosis is being made with greater frequency and earlier in the course of the disease. The main adjuncts to clinical diagnosis are special x-ray methods of visualizing the colon and pathological examination by rectal biopsy.

The treatment is surgical in all cases. In infancy, early colostomy (or ileostomy where indicated) may be life-saving, and is done as soon as the diagnosis is made. The pull-through operation is then carried out when the child reaches the required weight. In older children a primary pull-through operation may be performed if the general condition of the child is satisfactory.

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RÉSUMÉ

La maladie de Hirschsprung, ou mégacôlon congénital, est connue comme entité morbide depuis plus de 70 ans; ce n'est cependant que depuis ces quinze dernières années que l'on a des conceptions satisfaisantes sur son étiologie et son traitement. Hirschsprung a décrit en 1888 ses deux premiers cas et en collationnait 55 autres en 1905. Depuis, de très nombreuses publications ont été consacrées à cette question; une revue de la littérature est faite ici.

Trois théories furent avancées en vue d'expliquer l'augmentation de volume du gros intestin, à savoir: ou bien l'existence congénitale d'un intestin dilaté; ou bien un étranglement mécanique; ou bien enfin, des troubles dans l'innervation de l'organe. Plusieurs auteurs avaient trouvé et décrit l'existence, en aval du segment intestinal dilaté, d'une zone non dilatée dans laquelle les cellules nerveuses étaient étrangement rares aux endroits où auraient dû se voir les plexus myentériques. L'importance de cette constatation n'était pas apparue alors.

On estime qu'un cas de maladie de Hirschsprung se recontre par 20,000 ou 30,000 nouveaux-nés vivants. Le sexe masculin est plus touché, dans une proportion de 80 à 90%. Peut-être existe-t-il certains facteurs de prédisposition familiale. Cliniquement, il faut distinguer deux formes de l'affection:

1.—la maladie de Hirschsprung classique: les symptômes en sont une constipation chronique et une distension abdominale d'importance variable accompagnées de fréquents épisodes d'iléus ou de subiléus à répétition. Des masses fécales peuvent parfois être palpées à travers la paroi abdominale. Le plus souvent, ces enfants sont sous-développés et en médiocre état de nutrition. Les rayons-x montrent des anses dilatées, contenant des matières et des gaz. Le lavement baryté met en évidence un segment intestinal proximal dilaté auquel fait suite une portion plus rétrécie immobile, sans péristaltisme.

2.—la maladie de Hirschsprung chez le nouveau-né, qui fut décrite comme entité morbide distincte par Ehrenpreis. Dans les premiers jours de la vie, le méconium est difficilement expulsé; des vomissements apparaissent précocement, accompagnés de distension de l'abdomen. Les épisodes d'obstruction se succèdent et conduisent le patient à une mort rapide. Ici, le lavement baryté ne montre rien de très caractéristique, si ce n'est l'impossibilité de dilater le bas intestin lors du remplissage par la bouillie opaque.

Dans chacune de ces formes, une diarrhée peut apparaître, qui semble causée par une entérocolite secondaire. Le diagnostic ferme se fait lors de l'examen anatomo-pathologique, qui permettra de constater l'absence de cellules nerveuses dans les plexus myentériques. Il convient de signaler à ce propos que l'on a décrit une technique de biopsie de la paroi intestinale avec sa musculaire.

Le traitement de la maladie de Hirschsprung a été décevant jusqu'au moment où l'on est arrivé à la conception actuelle de son étiologie. Le traitement médical n'est applicable qu'aux cas bénins. Chirurgicalement, on a pratiqué jadis des sympathectomies lombaires et des amputations des segments dilatés. Les résultats n'étaient pas durables. En réalité, il faut amputer, outre le segment proximal dilaté, tout le segment distal apéristaltique jusqu'à l'anus, et ensuite abaisser l'intestin à l'anus. Chez les enfants de moins de 18 mois, il sera prudent d'établir une colostomie.

Les auteurs passent alors en revue 58 cas provenant de leur pratique à l'Hôpital des enfants malades de Toronto; sur ce chiffre, 46 sont des garçons. L'apparition des premiers symptômes remontait à une date variant de un jour à six ans; un tiers des malades était âgé d'environ une semaine et une moitié d'environ un mois. La mortalité globale fut de 37%. Un traitement chirurgical fut appliqué à 45 enfants et consista en une colostomie simple isolée, ou en une résection selon Mikulicz ou enfin en une résection suivie d'abaissement. Les autres furent traités médicalement, ou non traités, ou encore traités chirurgicalement, mais de façon inadéquate; le diagnostic ne fut parfois posé qu'après la mort.

En conclusion, les auteurs estiment que le meilleur traitement de la maladie di Hirschsprung est l'excision large de tout le segment dilaté et du segment non dilaté situé en aval jusqu'à l'anus, suivie de l'abaissement de l'intestin.

IDIOPATHIC PNEUMOPERITONEUM

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THE DISCOVERY of free air within the peritoneal cavity is generally associated with the signs and symptoms of peritoneal inflammation, and is regarded as an acute abdominal emergency. This situation is usually detected by the physician or surgeon using clinical methods, or by the radiologist with fluoroscopy or x-rays in a very ill patient. However, the detection of free air in the peritoneal space, in a person who has no severe symptoms or signs related to the abdomen and is obviously not in distress, is disturbing both to the radiologist and the clinician. Absence of liver dullness on percussion in such a patient is indeed perplexing when first encountered. When the presence of free air below the leaves of the diaphragm has been confirmed radiologically, and there is no external source to account for it, a diagnosis of spontaneous or idiopathic pneumoperitoneum should be considered. The following case history is an example.

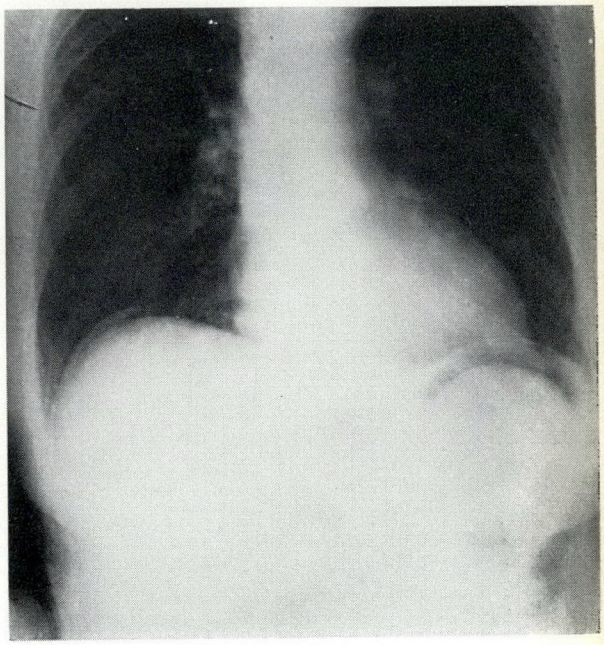

Fig. 1

CASE REPORT

CASE 1.—M.S., a 72 year old housewife, had been complaining of mild, intermittent, crampy abdominal pains for the past year and had lost 20 lb. In addition, she suffered from periodic epigastric fullness and flatulence, often associated with borborygmi. Her past history was non-contributory, apart from a very real emotional problem in her home life which disturbed her greatly whenever it was mentioned. She had had no previous abdominal operations.

Because of her weight loss and vague upper abdominal complaints, her physician arranged for her to have a gastric series as an out-patient. During the initial fluoroscopy, the radiologist was surprised to find free air underlying both leaves of the diaphragm (Fig. 1). This air was seen to shift when the patient was moved in order to take radiographs in the supine, erect and lateral recumbent positions (Fig. 2). Needless to say the plan for the administration of barium was cancelled, as it was thought that this patient had a perforated viscus.

On examining her abdomen, there was ample evidence of air under the right diaphragm, but no signs of enlargement of viscera, or other abnormal masses or tenderness could be elicited. The results of the remaining clinical examination were entirely normal including that of her neurological system, and her pain threshold was regarded as within normal limits. She was admitted to hospital for investigation.

The patient was confined to bed and given a bland diet as her only form of therapy. Her gastrointestinal tract continued to function

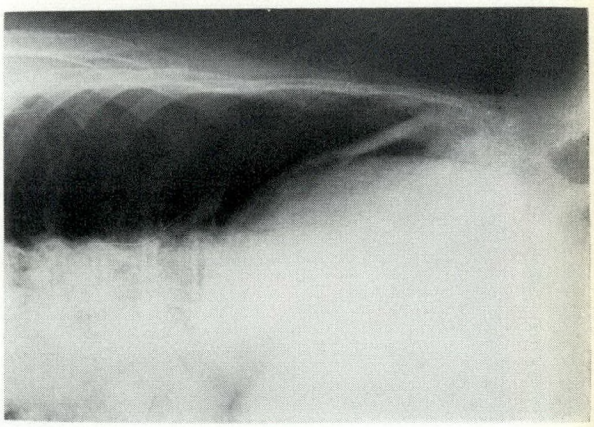

Fig. 2

°Wellesley Surgical Division, Toronto General Hospital.

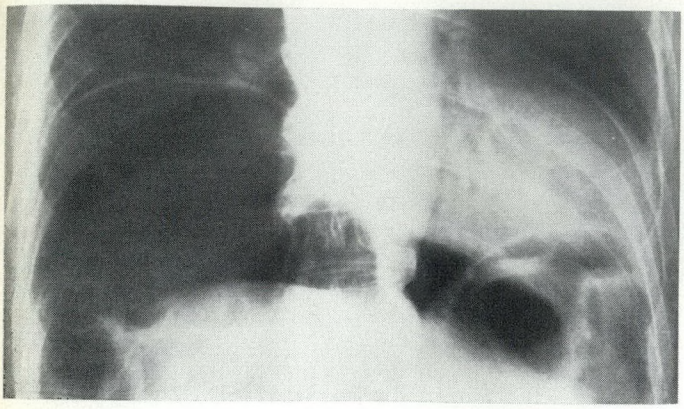

Fig. 3.

normally and she remained relatively asymptomatic.

Her hæmoglobin level and red and white blood cell counts were normal, as was her blood smear. The Wassermann reaction was negative and she had no fever. Results of rectal examination, proctoscopy and sigmoidoscopy were also normal.

At the end of a week, her clinical picture had not changed, although a decrease in the amount of free air was seen on further abdominal radiographs. It was thought that by then any leakage from a viscus would be sealed over, and a gastric series and motor meal examination was carried out. This showed no abnormalities in the œsophagus, stomach, pylorus or duodenal caput, but three or possibly four diverticula of the second part of the duodenum were seen. The barium in the small bowel was carefully observed as it passed onwards, and it was noted that the upper portion was dilated, and that there was an excessive amount of gas in it at the commencement of the examination. No further diverticula or other abnormalities were visualized, and the contrast medium passed on into the large bowel in a normal manner without delay. A subsequent gall bladder series and barium enema examination failed to show any peculiarities.

As this patient continued to remain well, she was discharged from hospital with a diagnosis of duodenal diverticula and idiopathic pneumoperitoneum.

Six months later she returned, complaining of a swelling in her right groin which she had observed as it slowly increased in size during the preceding two months. Since her previous admission to hospital, she had carried out her usual household duties, being bothered only occasionally by her original symptoms of epigastric fullness, flatulence and vague abdominal distress. Her weight remained stationary.

An easily reducible inguinal hernia with a posterior wall defect about three inches (7.5 cm.) in diameter was present. Once again, the presence of free air over the liver was demonstrated clinically but no other abnormalities were found in her abdomen.

She was subsequently admitted to hospital for repair of this gradually enlarging hernia, which had not bothered her in any other way. Abdominal radiographs (Figs. 3 and 4) showed free air in copious quantities under her diaphragm, with a space of four inches (10 cm.) between the right leaf and the upper surface of the liver. In addition, bowel was interposed.

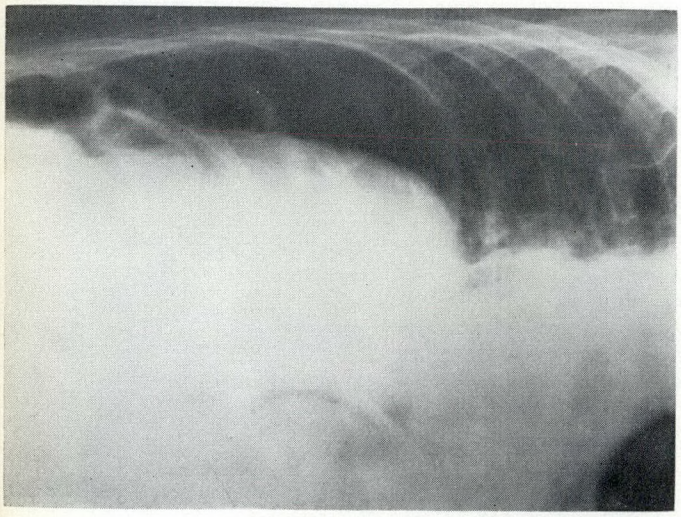

Fig. 4.

The radiologist also reported that many loops of small bowel were seen, some of which contained fluid levels. In addition, he believed that a small amount of free fluid was present in the peritoneal cavity. It was decided preoperatively that, if it was possible during the repair of her hernia, her peritoneal cavity would also be examined. This was subsequently done by extending the hernial incision in a more vertical direction than is customary. An opening was made into the large hernial sac, and immediately gas bubbled out through straw coloured fluid which was present. Altogether about 200 c.c. of the fluid was removed, which was sterile on culture and showed no cells in the smear. The hernial sac contained no viscera.

The right colon was easily delivered through the wound and no intraluminal lesion could be palpated. Its wall was of a normal thickness. The serosal surface of the cæcum and a small part of the ascending colon was excessively reddened in between stellate, greyish plaques which were thought at the time to be well organized fibrin deposits. The appendix was normal, as was the terminal ileum for a distance of four feet (1.20 m.). From here to the proximal three or four feet of the jejunum, the outer surface of the small bowel was plastered with the same stellate, pearly grey and slightly raised markings. They were considerably more numerous here than on the cæcum or ascending colon. These were distributed not only on the side walls of the bowel, but also on the peripheral portion of the mesentery on either side, and closely followed the paths of the blood vessels along the most distal arcades. The bowel wall was otherwise normal, as was the remaining portion of its mesentery. No enlarged lymph nodes were seen and a Meckel's diverticulum was not present. It was thought that it would be extremely difficult to remove these serosal plaques, as they were intimately adherent to the walls of the bowel and the mesentery, and a more radical approach was not thought to be advisable. This lesion did not in any way resemble Crohn's disease, ulcerative colitis, tuberculous enteritis, or any form of malignancy.

Fig. 5.

Because of a considerable degree of visceroptosis, the proximal four feet of jejunum was well visualized. Along the mesenteric side of this portion of bowel were multiple large diverticula (Fig. 5). None were present on the anti-mesenteric border and none showed any evidence of perforation. These diverticula were large. Some of them measured two to three inches (5-7.5 cm.) in all diameters, and they had large apertures in continuity with the lumen of the bowel.

When the bowel was compressed, fluid-gas levels could be clearly seen through the thin distended walls of the diverticula themselves, but no leakage of gas or fluid was observed. The diverticula were extremely numerous and resembled clusters of huge grapes, some of them being multiloculated and in open communication with each other. This portion of the small bowel and its mesentery were otherwise normal. The remaining abdominal and pelvic organs were also normal, but perhaps excessively mobile. It was thought unwise at the time to subject this patient to a bowel resection, because of doubt as to which portion should be removed and because her symptoms were minimal. In addition, she was 72 years old and we were actually operating on her for another condition. Consequently the inguinal hernia repair was carried out in standard fashion.

There were no postoperative complications and the patient left hospital 10 days afterwards.

Discussion

Free air in the peritoneal cavity is a common condition and may occur from a wide variety of lesions and causative agents, so that it is probably worth while to consider its origin in some detail. One must suppose that a rupture or leak of a gas-filled viscus or cyst has produced this entity, once the absence of any recent operative, therapeutic or diagnostic procedure has been established. Infection due to gas-producing bacteria is as a rule accompanied by the signs of peritonitis or abscess formation. It is of course extremely rare to encounter a patient with pneumoperitoneum not accompanied by peritonitis, but this does occur occasionally either with or without a demonstrable cause.

Therapeutic, diagnostic or operative procedures may leave residual gas in the peritoneal cavity. The intraperitoneal instillation of air into this space is still used in the treatment of certain cases of pulmonary tuberculosis.

Fallopian tube patency may be ascertained with air or other media under pressure and can produce pneumoperitoneum.

Rupture of a gas-containing viscus allowing the escape of its contents may follow penetrating abdominal or pelvic wounds, or instrumentation such as gastroscopy or sigmoidoscopy. These accidents are invariably accompanied by peritonitis and may be due to direct trauma of the wall of the viscus or to instillation of an excessive amount of air through the instrument. Loughead[1] cites an instance, however, of the development of pneumoperitoneum after an air contrast barium enema without the escape of barium from the bowel and without evidence of peritonitis.

After abdominal operations, it is usual for air to remain in the peritoneal cavity, but this rarely produces any symptoms and is usually completely absorbed in seven to ten days.

Various lesions of the bowel may directly or indirectly produce a perforation permitting an escape of gas, which is almost always accompanied by intestinal content, into the peritoneal space. In such an instance the signs of peritoneal irritation will be added to those of pneumoperitoneum. Rupture of a viscus from external trauma, or spontaneously from a pre-existing disease such as peptic ulcer, neoplasm of the gastrointestinal tract, or colonic diverticulitis, is probably the commonest reason for this condition. On rare occasions, only gas may leak out; this does not give rise to the signs and symptoms of peritoneal inflammation. A description of such a case follows:

Case 2.—J.T.H., an 83 year old man, suffered over a period of two months from epigastric pain and vomiting and lost 20 lb. A gastrointestinal series was arranged and at this examination free air was found under the right leaf of the diaphragm (Fig. 6). He was immediately sent into hospital although his only complaint was mild epigastric distress and his temperature, pulse rate and white blood cell count were normal. His abdomen was soft on palpation and no tenderness or masses were present, but liver dullness was absent on percussion. The results of the remaining examination were normal.

The following day, air was still present in the peritoneal cavity, and with constant gastric suction he remained well. It was thought that the free air was derived from a leak in a peptic ulcer or carcinoma of the stomach.

In a week, it became apparent that he was suffering from pyloric obstruction, not alleviated by continuance of gastric suction, and he was operated upon. Here a polypoid carcinoma of the gastric antrum was found and, because of his age, only a gastro-enterostomy was performed. There was no evidence of a perforation or peritonitis.

This is an instance of asymptomatic pneumoperitoneum, which may or may not be idiopathic. Nanson and Dragan[2] reported a case of symptomless pneumoperitoneum without peritonitis and suggested that the site of perforation was in a jejunal diverticulum. The patient was relieved by resection of the involved portion of small bowel. The same authors also drew attention to the fact that idiopathic pneumoperitoneum was infrequently mentioned in the medical literature, and referred to only 20 examples up to 1956. These cases may have an associated but undemonstrable lesion as the source of the free air; peritonitis is rarely associated with it.

Sidel and Wolbarsht[3] described a patient with pneumonia and an asymptomatic pneumoperitoneum that disappeared completely after a three-month interval. Other supradiaphragmatic lesions may be suspect, such as an emphysematous bulla in contact with a thin eventrated diaphragm, as Hinkel[4] reports. He also refers to the rare appearance of pneumoperitoneum secondarily to an artificially produced pneumothorax when the needle has been inserted into the pleural space only.

As far as can be ascertained, Ayres[5] first discussed the association of pneumoperitoneum and "gas cysts of the intestinal tract" in the literature in the English language. He translated from Stenstrom's[6] article on this subject, and pointed out that this lesion is apparently more common in Europe and is correctly known as pneumatosis cystoides intestinalis or emphysema bullosum intestinale. This disease is characterized by the formation of many cysts, which are generally located on the small intestine but may occur elsewhere in the gastrointestinal tract. Their etiology remains obscure.

Gazin *et al.*[7] comprehensively discussed this disease and suggested that, should the

cysts rupture and form a pneumoperitoneum, gas may be seen by x-rays outside the gastrointestinal tract. Other authors[8-10] describe this also, but as Koss[11] in his review in 1952 collected only 213 published cases of pneumatosis cystoides intestinalis, it must be a rare source of free gas in the peritoneal cavity, since rupture of the cysts does not by any means always occur. According to Ayres and Dale, this is often a self limiting disease and there may be a local inflammatory and foreign body reaction which progresses to a chronic stage with ultimate fibrosis and scarring. The diagnosis of this condition as the source of pneumoperitoneum may be well nigh impossible, for the cysts may entirely disappear with the passage of time, leaving small greyish-white plaques or fibrotic areas on the wall of the gut and neighbouring mesentery. This observation well describes the lesions that we encountered on the serosal surface of the bowel in our first case.

The case of a patient with recurring attacks of epigastric distress, abdominal distension and vomiting associated with a degree of pyloric obstruction and pneumoperitoneum has been well described by Trachtman and his colleagues.[12] At operation, no lesion other than an area of patchy fibrosis of a segment of small bowel wall was present. They concluded that this was an instance of pneumatosis cystoides intestinalis and that this was the source of the free gas. At no time during the four and a half years that this case was followed up were there any symptoms or signs of peritonitis, but some degree of pneumoperitoneum was always present. It is even more disconcerting, as Schneewind[13] has intimated in his description of a patient with pneumoperitoneum and evidence of peritoneal irritation upon whom he operated, to discover no abnormalities at all. A subsequent radiological investigation showed a normal gastrointestinal tract.

Summary

A case of persisting pneumoperitoneum has been presented, in which no signs or symptoms of peritoneal inflammation were present, and no significant abnormalities detected on a radiological investigation of the gastrointestinal system. Six months later, while an inguinal hernia was being repaired on this patient, an opportunity to examine the abdominal and pelvic organs became available. Free gas and a small amount of clear yellow fluid were present in the peritoneal cavity. Marked jejunal diverticulosis, in addition to a patchy fibrosis of a portion of small bowel wall and adjacent mesentery, was also found. The patient's initial symptoms of epigastric distress, flatulence and borborygmi could well be accounted for by the jejunal diverticula, because in this condition, the roentgenological diagnosis is often difficult.

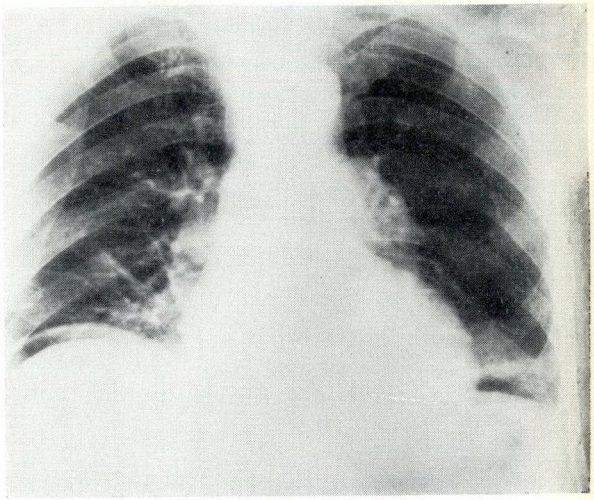

Fig. 6.

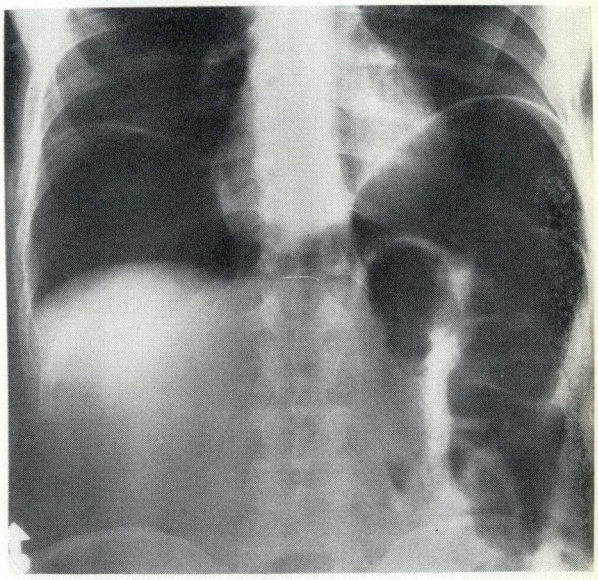

Fig. 7.

There was no evidence of peritonitis or perforation, however, to account for the free gas in the peritoneal cavity. It is possible that, because of the thin translucent walls of the jejunal diverticula, gas may have diffused through them into the peritoneal space without producing a gross site of leakage. The stellate shaped, pearly grey plaques seen over the serosal surface of a segment of ileum and jejunum and their mesentery were suggestive of the fibrotic end stage of pneumatosis cystoides intestinalis. Several authors have indicated that this condition, even at this stage, may cause pneumoperitoneum.

Unless this patient's peritoneum does not allow the free transmission of gas, which is unlikely, then a continuing supply is being produced from some source that is suspected but unproven at this time.

Addendum

Since this paper was written, another case of pneumoperitoneum of unknown origin has been encountered. In this instance, a 60 year old male patient developed epigastric pain of moderate severity two weeks before he was admitted to hospital. During this period he was always able to consume his normal diet without any distress, and the pain gradually abated. He had no other symptoms and his bowels moved normally. On admission to hospital he complained only of very slight discomfort in the left side of his abdomen, where slight tenderness was also present. His abdomen was distended and tympanitic, and liver dullness was absent. No other abnormal findings were noted clinically, but a radiograph showed a large collection of gas under both sides of his diaphragm (Fig. 7).

With conservative management only, his symptoms rapidly disappeared and results of a gastrointestinal series, barium enema and sigmoidoscopic examination were normal. At the time of discharge three weeks after his admission, he was entirely asymptomatic and the pneumoperitoneum of undisclosed origin had entirely disappeared.

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Résumé

La présence d'air dans la cavité péritonéale est habituellement considérée comme le signe d'un trouble grave. Cependant, il peut s'agir d'un pneumopéritoine essentiel à évolution bénigne comme l'illustre le cas d'une femme de 72 ans rapporté par l'auteur. Cette malade accusait des coliques depuis un an et une perte de poids de 9 kilos. A la fluoroscopie on découvrit la présence d'air sous le diaphragme. L'examen clinique à part la disparition de la matité hépatique ne révéla rien qui put expliquer cette anomalie. Elle fut mise en observation avec diète légère mais la fonction intestinale n'en demeura pas moins normale. Après une semaine on se risqua à lui administrer un repas baryté qui montra trois diverticules de la seconde portion du duodénum.

Six mois plus tard elle se présenta de nouveau pour une masse dans l'aine qui était une hernie inguinale droite. Non seulement on nota encore la présence d'air libre dans la cavité péritonéale mais aussi une aéro-hydro-iléie segmentaire. A l'opération, dès que le péritoine fut ouvert on sentit barbotter des bulles d'air qui s'échappaient, et l'on retira environ 200 c.c. d'un liquide jaunâtre et stérile. A environ 1.2 m. de l'iléon terminal, le grêle était couvert de points gris perle en astérisques qui représentaient autant de petites cicatrices et que l'on voyait aussi sur le mésentère le long des arcades vasculaires. Le début du jéjunum comportait plusieurs gros diverticules en grappes. Après contention de la hernie on referma l'abdomen et les suites postopératoires furent sans histoire. Un autre cas semblable chez un homme de 60 ans est aussi rapporté en addendum.

A part les cas où le pneumopéritoine est créé comme mesure thérapeutique ou diagnostique, la présence d'air ou de gaz dans le grand ventre est le résultat de perforations traumatiques ou pathologiques, des suites fâcheuses d'une instrumenta-

tion malheureuse, ou d'un ensemencement bacillaire. Dans la grande majorité de ces cas on observe des signes non équivoques de péritonite. On prétend que certaines perforations ne permettraient que l'échappement de gaz. Il semble que le pneumopéritoine essentiel soit le résultat de la perméabilité des vésicules de la pneumatose kystique.

MODERN TRENDS IN GASTROENTEROLOGY (Second Series). Edited by F. Avery Jones, Central Middlesex Hospital, London. 415 pp. Illust. Butterworth & Co. (Canada) Limited, Toronto, 1958. $15.75.

The first volume of this series appeared in 1952 and contained 34 chapters and more than 800 pages. Although it too attempted to emphasize recent advances in selected subjects it dealt with many subjects in a much more general way. The present volume is designated by Illingworth in his introduction as a companion volume—a most apt description of this excellently produced, beautifully planned, compact book, which brings one up to date in all the important fields of gastroenterology. It is not a textbook on the subject and systematic treatment of gastroenterology is left to such standard books as are available, but every one of the 24 chapters contains the most up-to-date information on its particular subject and a very full list of references.

It is difficult to single out any particular feature but the reviewer feels that the inclusion of American experts such as Bockus and Palmer and of writers from other countries has given this book an international flavour and enhanced the value of its contents, as being of global validity. Jan Waldenström of the University of Lund gives a lively description of the clinical picture of carcinoid tumours; Bengt Pernow of the Department of Physiology at Stockholm, deals with the chemical and physiological aspects of these tumours, while the pathologist Basil C. Morson of London, England, gives a fine description of the general and special pathology of carcinoid tumours including their method of spread.

The chapter on steatorrhœa by W. T. Cooke and J. M. French is typical of most of the chapters on other clinical conditions. It is written by experts who have themselves made important contributions to the subject and are able to present it lucidly and convincingly.

Anyone familiar with the textbook and the numerous writings of H. L. Bockus will enjoy the chapter on ulcerative colitis. The effects of ACTH and corticosteroids on the alimentary tract is a timely topic and is covered by L. J. Witts of Oxford. He deals with the undesirable effects on the alimentary tract in the therapeutic use of these agents as well as their use in the treatment of hepatitis, steatorrhœa and of course ulcerative colitis.

The chapters on the Mallory-Weiss syndrome, the Peutz-Jeghers syndrome and newer knowledge of the physiology of smooth muscle of the alimentary tract were particularly instructive to this reviewer. The comprehensive approach to cancer of the alimentary tract will serve as a guide for many writers on the subject for years to come. The publishers can be congratulated on this well produced book and on the excellent illustrations.

AN INTRODUCTION TO EXPERIMENTAL SURGICAL STUDIES. W. J. Dempster. 463 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1957. $12.00.

Dempster's book is intended for the postgraduate student of surgery first engaging in experimental surgery and research.

The method of approach is by selection of a clinical problem followed by an outline of the general trend of research in that particular field, including the basic surgical fundamentals essential in considering and developing the problem.

The selection of topics is excellent, particular attention being given to those problems of current interest. Tissue regeneration, wound healing, tissue grafting and transplantation are adequately covered. The problems of hypothermia and hypertension are of special interest, as is the consideration of the cardiovascular system dealing with cardiac surgery, heart-lung substitutes, shock and peripheral vascular phenomena.

Chapters 2 to 4 are concerned with the behaviour of injured tissues under various conditions. The biochemical data are well documented and present an adequate introduction to the special problems which come later.

Of particularly high quality is the section on genito-urinary investigation which aptly displays Dempster's enthusiasm and original work in this field. The section on gastroenterological problems is short; however, it contains a tremendous amount of experimental data on stomach, intestine, pancreas and liver, and the presentation is excellent. Reference selection and documentation are excellent throughout the book.

This book is a valuable contribution for every research laboratory. The book should be read not only by the postgraduate student of surgery, but by every surgeon who would like to give his patient the benefit of the advances of his profession and at the same time protect the patient from unwarranted experimentation.

CIRCULATING FIBRINOLYSIN IN CARCINOMA OF THE PROSTATE

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FIBRINOLYSIS IS THE aseptic breakdown of whole blood clot. The first reported case of this phenomenon associated with metastatic carcinoma of the prostate was that reported by Tagnon and his colleagues[1] in 1952. Since then, there have been numerous case reports in the literature.[2-4] While similar types of reaction have been reported in association with pregnancy, overwhelming infection, benign prostatic hypertrophy and several types of carcinoma, this presentation deals only with that associated with metastatic carcinoma of the prostate.

Huggins[5] first demonstrated the presence of fibrinolysin in normal prostatic tissue and Tagnon showed it to be present in carcinomatous prostatic tissue. This latter fact has been confirmed by Aboulker.[6] There are at present two theories as to the possible mechanism of the fibrinolysis in carcinoma of the prostate. The first is that held by Tagnon: "That the fibrinolysin is produced by the prostatic tissue and acts directly on either the fibrinogen or fibrin to dissolve the clot". The second, that held by many hæmatologists, is: "That the products of the carcinoma act on an activator present in the blood and this activator acts on profibrinolysin which is normally present to produce fibrinolysin; the latter in turn acts on the fibrinogen to dissolve the clot". This fibrinolysin has been found present in 12% of cases of carcinoma of the prostate with metastases.[2]

This paper is based on six cases of carcinoma of the prostate seen at the Vancouver General Hospital and Shaughnessy Hospital in a one-year period. All were cases of widespread metastatic disease and all showed the presence and effects of circulating fibrinolysins.

CASE 1.—E.S., a 57 year old man, was first seen in March 1955, when he was admitted to hospital with gross hæmaturia and clot retention. Carcinoma of the prostate had been diagnosed in 1954 and treated by orchiectomy and stilbœstrol. X-ray therapy to the spine for metastatic lesions had also been given in 1954.

On admission to hospital, the non-protein nitrogen (N.P.N.) was 42 mg. %, the serum acid phosphatase was 2.3 King-Armstrong units and the alkaline phosphatase was 11 K.A. units per 100 ml. Prothrombin activity was 100% of normal. Radiographs of the spine and pelvis showed evidence of widespread sclerotic lesions. Clots were evacuated from the bladder and 6 g. of adenocarcinomatous tissue was removed by transurethral resection. Drainage was clear at the end of the procedure, but during the next 24 hours the bladder had to be evacuated of stringy clots three times. A total of 6000 c.c. of whole blood was given.

The test for circulating fibrinolysin was positive. Stilbœstrol therapy having been ineffective, the patient was started on cortisone 25 mg. four times a day. The result was dramatic; bleeding was controlled after the second tablet and did not recur during the next three weeks. This patient died from an acute coronary thrombosis at that time.

CASE 2.—T.L., a 72 year old man, was admitted to hospital on April 2, 1955, complaining of difficulty in walking and inability to void. The N.P.N. was 39 mg. %, acid phosphatase 82 K.A. units, alkaline phosphatase 11 K.A. units per 100 ml. A transurethral resection was done on April 4, and a diagnosis of carcinoma of the prostate was confirmed. Excessive bleeding followed the operation and he required a total of 4500 c.c. of blood during the next week. The prothrombin activity was 50% of normal and showed very little response to vitamin K.

The presence of circulating fibrinolysins was proven and the patient was started on cortisone, 50 mg. intramuscularly twice a day and stilbœstrol, 5 mg. three times a day intramuscularly and orally. During the following week, bleeding was completely controlled and the acid phosphatase level dropped to 4 K.A. units per 100 ml.

This patient later had a decompression of the dorsal spine for relief of pressure from secondary carcinomatous deposits with no difficulty from bleeding. He died shortly after this of a bronchopneumonia.

CASE 3.—T.C., an elderly patient, was admitted to hospital with a history of gross

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hæmaturia for an indefinite period of time. He was mentally confused and never able to give a significant history. On physical examination diffuse areas of ecchymosis were seen over the entire thorax and abdomen, as well as on the extremities. Rectally, the prostate was stony hard and fixed. The hæmoglobin level on admission was 80%, N.P.N. 47 mg. %, acid phosphatase 46.8 K.A. units and alkaline phosphatase 64 K.A. units per 100 ml. The urine grew no microorganisms on routine culture. At cystoscopy some gross blood was seen coming out from each ureteral orifice and the prostate bled freely. Retrograde pyelograms were normal. Radiographs of the spine and pelvis revealed extensive sclerotic lesions compatible with secondary metastatic carcinoma of the prostate. A complete hæmatological examination was done and circulating fibrinolysins were found. The platelet count was normal. The prothrombin activity was 65% of normal. Because the hæmorrhagic manifestations were increasing and the hæmoglobin level was dropping, combined methods of recommended treatment were instituted. The patient was given prednisone (Meticorten) 10 mg. four times a day, blood transfusions (a total of 3500 c.c.), and one bottle of serum albumin. Intravenous stilbœstrol diphosphate (Honvol) was administered in daily doses of 500 mg. The hæmaturia subsided rapidly and the areas of ecchymosis began to decrease although the blood still showed evidence of large quantities of fibrinolysins. Eleven days later a biopsy specimen of bone from the right iliac crest showed carcinomatous cells staining strongly positive for acid phosphatase by Gömöri's method. One week later, the intravenous stilbœstrol was discontinued and the patient was given the oral preparation.

Although he had shown no untoward immediate response to the bone biopsy, within two days after discontinuance of the intravenous preparation the gross hæmaturia began again and there was diffuse subcutaneous hæmorrhage at the site of the biopsy and extending down into the scrotum. Intravenous stilbœstrol was again started and continued for a further two weeks, at which time the patient was so well that he refused the intravenous injection and was given oral stilbœstrol, 5 mg. three times a day. While he was on the intravenous preparation, his acid phosphatase fell from the highest recording of 46.8 K.A. units gradually but progressively to 11.2 K.A. units per 100 ml. on the date of discontinuing the intravenous preparation. It continued to fall while he was on the oral preparation to a low point of 3 K.A. units. There was no change in the alkaline phosphatase level, which remained markedly elevated. Hæmorrhagic manifestations did not recur but the blood continued to show small amounts of circulating fibrinolysins. Repeat radiographs of the lumbar spine and pelvis showed no demonstrable changes in the appearance of the metastases. The patient remained in good health for six months, after which he was lost to sight.

CASE 4.—W.G., an elderly man, was first admitted to hospital in December 1951, with urinary obstruction. The acid phosphatase level was 20.7 K.A. units. Radiographs of the spine and pelvis revealed multiple osteoblastic metastatic lesions. Twenty-eight grams of prostatic tissue was resected, and the tissue showed an adenocarcinoma apparently well differentiated. The patient was discharged on stilbœstrol therapy. He got along reasonably well until 18 months before his recent admission to hospital, when symptoms of increasing urinary obstruction began. The patient learned to catheterize himself and apparently did this for several months. Eventually he required periodic urethral dilatations. He was admitted to hospital on September 28, 1955, with a history of increasing weight loss and periodic gross hæmaturia in addition to his obstructive uropathy. Acid phosphatase level on admission was 20 K.A. units; alkaline phosphatase 12 K.A. units per 100 ml.; N.P.N. 34 mg. %; hæmoglobin level 75%. An intravenous pyelogram showed a hydronephrosis of the left kidney and some dilatation of the right ureter. Radiographs of the spine and pelvis showed the presence of osteoblastic metastases. On October 1, 1955, 17 g. of prostatic tissue was resected. The procedure took 40 minutes and he appeared to withstand it well. Blood pressure was maintained at 100/80 mm. Hg. Drainage on the afternoon of operation was only moderately blood-tinged. At 3.00 a.m. on October 22, the patient's blood pressure dropped to a systolic level of 70 mm. Hg and urethral drainage was found to be brightly sanguineous. There was a considerable amount of bleeding around the catheter but clots were not a problem. The presence of circulating fibrinolysins was proven. Bleeding was temporarily controlled by traction on the urethral catheter. From then until his death, the patient continued to bleed heavily at intervals, and his blood pressure could not be maintained within normal limits. A total of 3000 c.c. of blood was administered without effect, although the hæmoglobin level was never allowed to drop below 63%, with a

hæmatocrit of 27%. The patient died in shock the following day.

At autopsy, the immediate cause of death was thought to be pulmonary œdema. There were generalized metastases from the carcinoma of the prostate.

CASE 5.—In March 1954, W.W. was admitted to hospital with a history of urinary obstruction. Examination revealed a large prostate. On March 20, a retropubic prostatectomy was performed. At this time it was obvious to the operator that either a malignant tumour or an infarction was present, and the bulk of the prostate was enucleated. The weight of the enucleated prostate was 47 g., and microscopically it showed the picture of benign nodular hyperplasia. On March 19, the acid phosphatase level was reported as 12.7 K.A. units per 100 ml., and on March 27, after operation, it was recorded as 1.8 K.A. units. Results of other laboratory tests were otherwise within normal limits.

After discharge from hospital, the patient continued to have gross hæmaturia and dysuria. He was re-admitted to hospital on May 30. On June 19, 15 g. of prostatic tissue was resected, and a widespread poorly differentiated adenocarcinoma was found in this. Acid phosphatase level was recorded as 4.8 K.A. units per 100 ml. on that admission. He was discharged on July 8, 1954, on oral stilbœstrol therapy.

The patient was not re-admitted to hospital until September 2, 1955. He had had a course of deep x-ray therapy in the meantime. On his final admission to hospital, the patient had gross hæmaturia with clots, which had become progressively worse. His hæmoglobin level on admission was 42% (6 g. %); his N.P.N. was 36 mg. %, acid phosphatase 5.7 K.A. units and alkaline phosphatase 20 K.A. units per 100 ml. The patient continued to bleed profusely, requiring evacuation of clots on several occasions during the 10 days before his death. The presence of circulating fibrinolysins was proven. The urine did become clear on occasions for a few hours, only to be followed by a repeated hæmorrhage. A total of 3600 c.c. of blood was administered. The highest recorded hæmoglobin reading during admission was 64%, accompanied by a hæmatocrit of 28%. Twenty-five mg. of cortisone was given twice daily without apparent effect. He died on September 12 in a terminal state of shock.

CASE 6.—V.T., a 77 year old man, was found to have a carcinoma of the prostate in 1951. He refused orchiectomy and had taken stilbœstrol intermittently since that time. Radiography of his spine showed metastatic carcinoma in the 11th dorsal vertebra.

He was re-admitted to hospital on January 23, 1956, because of excessive bleeding from the nose, from a small cut on the face, and from his mouth. A small puncture wound in his left ear poured blood. His retina showed evidence of hæmorrhage; his urine was grossly bloody and stools were positive for blood.

The prothrombin was less than 10% of normal; the acid phosphatase level was 25 K.A. units and the alkaline phosphatase level was 58 K.A. units. The presence of circulating fibrinolysins was proven and treatment was started with intravenous stilbœstrol diphosphate (Honvol), 250 mg. the first day and 500 mg. on four succeeding days. This was followed by stilbœstrol 5 mg. three times a day orally. Bleeding was completely controlled within four days and his prothrombin activity was up to 100% in six days. A total of 2500 c.c. of whole blood had also been used. One month later his acid phosphatase level was down to 4 K.A. units.

DISCUSSION

Solomon and Stefanini[7] have suggested the use of cortisone in fibrinolytic states. Cortisone appeared to work dramatically in Case 1, but failed to influence the clinical picture in the other three cases in which it was used. It has been observed by Tagnon and Dolaz that there is no correlation between the level of acid phosphatase in the blood and the presence of fibrinolysin. Since the acid phosphatase level is raised only in 18% of the cases of metastatic carcinoma of the prostate and fibrinolysins occur in 12%, it is unlikely that any correlation will be found. The response to therapy, however, in Cases 3 and 6 seems to show a correlation between a declining acid phosphatase level and a decreased titre of fibrinolysin in the blood.

The prothrombin activity is almost always reduced in the presence of pathological levels of fibrinolysin. In Case 1 it was reported as 100% of normal but since this was the only reading there may have been a laboratory error. The reduction of prothrombin activity is probably due to the proteolytic effect of the fibrinolysin on the "prothrombin complex" substances.

Treatment

We are now on the alert for the presence of fibrinolysin in the blood of all patients we suspect of having carcinoma of the prostate. All these patients are screened by examining samples of blood after the method of Biggs and Macfarlane.[8] If a positive test is obtained, treatment is as follows:

1. No traumatic procedures such as catheterization or dilatation are allowed.
2. Intravenous stilbœstrol diphosphate (Honvol), 500 mg., is given daily.
3. Cortisone, 25 mg., is given four times a day.
4. Transfusions with packed cells are used. Packed cells are preferred because giving whole blood may mean pouring more fibrinolysins into the blood plasma.
5. Serum albumin contains an anti-fibrinolysin; while it had little apparent effect in the one case in which we used it, it may still be of some value.
6. Fibrinogen, 6 g., intravenously, is given at once and repeated if necessary. Fibrinogen is found in Cohn's fraction I. Since profibrinolysin also precipitates in this fraction with some of the methods, a suitable method for obtaining fibrinogen free of profibrinolysin must be used.
7. Blood is checked daily for the presence of fibrinolysin, as above.[8]

Summary

Six cases of carcinoma of the prostate with circulating fibrinolysin have been presented. Since it occurs in 12% of cases of carcinoma of the prostate with metastases, blood samples should be routinely screened in all cases. The value of various modes of treatment is discussed. Fibrinolysin from an unsuspected carcinoma of the prostate should be considered as a possible cause of any obscure bleeding problem in elderly males.

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Résumé

On parle de fibrinolyse lorsqu'il y a destruction aseptique d'un caillot sanguin. Ce phénomène survient dans certaines affections ou états, tels la grossesse, divers cas de carcinomatose et dans l'hypertrophie prostatique. Le présent article traite uniquement de la fibrinolyse que l'on observe dans des cas de cancer de la prostate.

On a pu mettre en évidence, dans le tissu prostatique normal, une enzyme responsable de cette action: la fibrinolysine. Plusieurs hypothèses ont été avancées pour expliquer ce mécanisme chimique: ou bien, la fibrinolysine serait capable d'agir directement sur la fibrine ou le fibrinogène; ou bien, le carcinome prostatique sécréterait un activateur de la profibrinolysine normalement présente dans le plasma, ce qui augmenterait le taux de fibrinolysine.

L'auteur présente alors, de façon détaillée, six observations personnelles de cas: chez tous, il s'agit de malades atteints de carcinome de la prostate ayant fait des hématuries spontanées et des hémorragies post-opératoires causées par une élévation anormale du taux de fibrinolysine (cette dernière fut dosée au laboratoire).

Le traitement de ce syndrome consiste à prévenir tout traumatisme: cathétérisations et dilatations urétrales devront être interdites; administrer du stilbœstrol et de la cortisone; transfuser, en cas de besoin, non avec du sang total, ce qui risquerait d'amener encore plus de profibrinolysine, mais avec des globules déplasmatisés; donner de la sérumalbumine qui est antagoniste de la fibrinolysine (bien que l'auteur signale n'avoir pas eu de résultat net dans le seul cas où il eut l'occasion de l'essayer); donner du fibrinogène; contrôler journellement par le laboratoire le taux sanguin de fibrinolysine.

POSTOPERATIVE BLEEDING AFTER EXTRACORPOREAL CIRCULATION*

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UNCONTROLLED GENERAL BLEEDING after surgery is becoming more frequent with extensive surgical procedures requiring massive blood replacement and in prolonged cardiovascular procedures involving extracorporeal circulation. During an extracorporeal procedure blood is subjected to an anticoagulant, an anticoagulant antagonizer and contact with such foreign surfaces as plastic, silicone and antifoam. Despite adequate hæmostasis during thoracotomy closure, postoperative hæmorrhagic episodes have been attributed to the following causes: deficiency of ionized calcium, elevation of citrate levels, platelet deficiency, fibrinolysis, depression of fibrinogen levels, decrease in factor V, incompatible blood transfusions, hypothermia and heparin rebound. One or two of the above mentioned factors have been carefully studied in patients with unusual bleeding, and when a change has been observed it has often been considered the cause of the bleeding episode. The purpose of the present study was to evaluate some of the commonly recognized blood factors responsible for clotting and control of hæmorrhage. It was carried out on clinical patients and laboratory animals, who were subjected to the extracorporeal circulatory system for varying lengths of time.

This study involves the changes found in platelets, prothrombin, fibrinogen and antihæmophilic globulin, during the time that the patient or laboratory animal was connected with the extracorporeal system. The results were compared with those in blood allowed to coagulate slowly in siliconed containers. This study was undertaken with full realization of the complexity of the coagulation mechanism, particularly when materials have been added to blood.

MATERIALS AND METHODS

Fourteen clinical cases were studied on the extracorporeal circulation, which consisted of the Cowan pump[1] and the DeWall bubble oxygenator,[2] and 14 laboratory animals were subjected to the use of an autogenous lung preparation with the Sigmamotor pump. Both the animals and the patients received heparin 1.5 mg. per kg. of body weight. The blood used to fill the extracorporeal circuit contained 1.8 mg. of heparin per 500 mg. of blood.

Methods

1. Whole blood clotting time (Lee and White[3]).

2. Platelet count (Brecher and Cronkite[4]).

3. One stage prothrombin (Quick[5]).

4. Two stage prothrombin area method (Biggs and Macfarlane[6]).

5. Thrombin – fibrinogen dilution curve (Biggs and Macfarlane[7]).

6. Thromboplastin generation test (Biggs and Douglas[8]).

7. Antihæmophilic globulin assay (Mustard's[9] modification of Pitney[10]).

8. Acid precipitated globulin determination (Douglas and Biggs[11]).

9. Thromboplastin dilution curve (Biggs and Macfarlane[12]).

10. *Fibrinogen determination.*—Ten millilitres of blood was withdrawn from the extracorporeal system and the cells were separated by centrifuging; to this plasma sufficient protamine sulfate was added to clot. The clot was then spun out on a weighed glass rod, washed twice in normal saline and once in distilled water, dried in an oven at 100° C. for one hour and then re-weighed to determine the amount of fibrin deposited.

11. *Clotting of blood in siliconed containers.*—One hundred millilitres of blood was taken from the external jugular vein

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of an animal by inserting a siliconed polyethylene tube into the right auricle and the blood was allowed to flow into a siliconed beaker. Three-millilitre samples were taken from the beaker every minute and placed in siliconed test tubes to which heparin (4 international units per c.c. of blood) had been added. These samples were taken until the blood clotted.

Study of the Blood Factors

Fibrinogen

Samples of blood were taken every five minutes during the extracorporeal procedure. Fibrinogen levels before perfusion ranged from 541 to 642 mg. % with an average of 580 mg. %, while the levels after perfusion ranged from 329 to 367 mg. % with an average of 352 mg. % after 40 minutes on the extracorporeal circulatory system. This was a drop of 52.8% in the amount of circulating fibrinogen. Blood which has been allowed to clot slowly in siliconed containers shows a similar decrease of 55.9%. The blood in the siliconed containers shows a more rapid fall than the blood in the extracorporeal system, because the blood in the latter is heparinized and clots at a much slower rate.

The minimum blood fibrinogen level is not easy to define. In an uncomplicated case of afibrinogenæmia it seems that 60 mg. % of fibrinogen is sufficient to restore normal clotting. It would appear from our studies that the drop in fibrinogen level is not severe enough in itself to be the cause of serious postoperative bleeding.

Prothrombin

The sample of blood taken at the beginning of the procedure was taken to be 100%, and to this all references were made. The results on the extracorporeal system show an initial increase in prothrombin during the time on the pump, reaching a height of 172% at the end of 20 minutes. In the siliconed beaker technique a peak of 240% was reached in two minutes. This is an "apparent" prothrombin increase, because in the method used one is actually measuring the yield of prothrombin generated and assuming that this represents the amount of converted prothrombin. It is

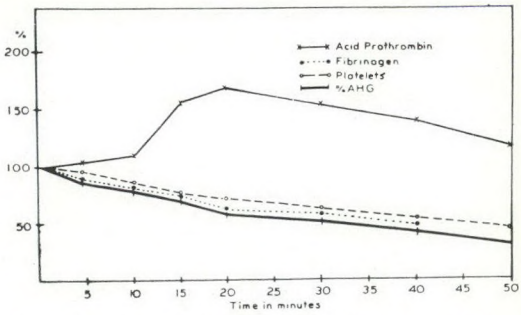

Fig. 1.—Composite blood picture during perfusion.

not clear whether this increase is due to the activation of a prothrombin "precursor", as was suggested by Bordet[13] and Quick,[14] or to the activation of a prothrombin "converter" which in turn increases the yield of thrombin from prothrombin, thus confirming the work of Ware and Seegers.[15] Both samples of blood show a decrease in prothrombin after these peaks, so that after 50 minutes of extracorporeal circulation and after 20 minutes in a siliconed beaker the blood showed a prothrombin level of 123%. In a small number of cases these results were confirmed by the area method of Biggs and Macfarlane.[16] The descending arm of the prothrombin curves in Figs. 1 and 2 parallel the other fractions which were being measured.

Since 50% or over is required for clotting it seems reasonable to conclude that the prothrombin is adequate for clotting and is not a cause of postoperative bleeding.

Platelets

The exact manner in which platelets function in blood coagulation and clot

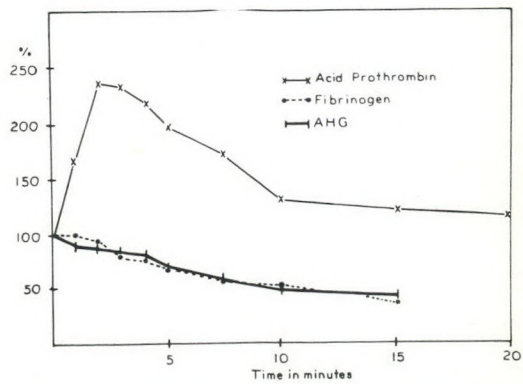

Fig. 2.—Composite blood picture—clotting in siliconed glassware.

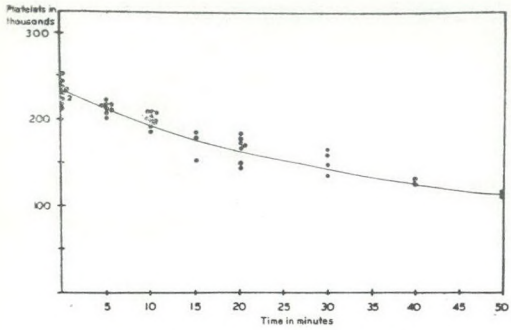

Fig. 3.—Platelets (in thousands).

retraction is not quite clear. The platelet count at the beginning of the operation on the laboratory animals varied between 215,000 and 251,000. During the procedure there was a decrease from an average of 231,000 to 116,000 over a period of 50 minutes. While the children were being perfused there was a drop from 377,000 platelets to an average of 152,000 platelets after they had been on the extracorporeal circuit for 20 minutes.

It was shown by Clifton,[17] and Ellis[18] and lately by Howland[19] that during operation and postoperatively platelets decrease in number. Ellis noted that after subjecting a dog to open cardiac surgery with occlusion of the great vessels the platelet count decreased on an average by 48,000. It is known that in cases of thrombocytopenia (especially secondary) there is a rough correlation between the platelet count and the severity of bleeding. Bleeding does not usually occur unless the platelets are fewer than 60,000 per c.mm. In the clinical cases which did bleed postoperatively it is noted that the platelets did not approach this level (see Fig. 3). Thus one might assume that the decrease in the number of platelets is not the cause for the bleeding tendency.

Antihæmophilic Globulin

The antihæmophilic globulin (AHG) level at the beginning of the operation on patients and animals was 100%. Samples were taken every five minutes while the subject was on the extracorporeal system and the AHG level was found to decrease to as low as 35% after 50 minutes of perfusion. The usual length of time of laboratory perfusion with the Sigmamotor pump and autogenous lung preparation was 30 minutes and the level of AHG varied from 45 to 64%. The lowest AHG levels were seen in an eight year old and a 16 year old patient on the pump for 56 and 48 minutes respectively, whereas the highest level was 70%—recorded on a seven month old and a six year old who were perfused for 30 minutes and 20 minutes respectively. It appears that AHG depletion is directly proportional to the volume flow and time of perfusion.

TABLE I.—Antihæmophilic Globulin Assay of Patients*

Patient	*Age*	*% AHG at end of perfusion*	*Time on pump*	*Post-perfusion bleeding*		
				O.R.	*R.R.*	*Total*
D.J.R.	7 months	70	30 min.	900	300	1200
D.M.	6 years	70	20 "	575	1000	1575
N.A.H.	13 "	60	24 "	1100	800	1900
T.C.	7 "	60	40 "	1100	1700	2800
J.B.	8 "	60	23 "	900	800	1700
G.G.	13 "	58	30 "	600	1000	1600
R.S.	6 "	55	28 "	1600	1100	2700
S.D.	10 "	55	30 "	1500	1400	2900
N.A.	10 "	50	50 "	1000	500	1500
J.F.	13 "	45	30 "	3700	900	4600
P.D.	9 "	45	20 "	2500	3200	5700
P.R.	12 "	45	36 "	2100	1500	3600
E.M.	8 "	23	56 "	3700	3000	6700
R.M.	16 "	23	48 "	1000	7000	8000

*Only patients who bled excessively after perfusion were included in this study.
O.R. = Operating room.
R.R. = Recovery room.

Discussion

From the observations of Brinkhous *et al.*,[20] on hæmophilic dog blood and Biggs and Macfarlane[21] on a series of patients investigated by the AHG assay method, it has been suggested that a level of antihæmophilic globulin of at least 35% of normal is required to maintain adequate hæmostasis and to prevent bleeding from minor injuries and that 50% is required to cover major injuries or operations. Hæmophilic patients with less than 20% AHG have bled severely, even though the clotting time and prothrombin consumption were normal. Below 10% AHG the prothrombin consumption is usually abnormal but it is not until the level is below 2-5% that the clotting time is prolonged.

Patients who bleed postoperatively may have a normal prothrombin consumption index and clotting time and yet have antihæmophilic globulin levels in the bleeding range.

If the AHG has been depleted below 50% by a long extracorporeal perfusion, it must be raised well above this level to restore clotting. The administration of large quantities of fresh whole blood as an empirical treatment for such postoperative bleeding probably brings about clotting by raising the AHG level. If this is true, and we believe it is, it would be wise, particularly in children, to determine the AHG level at the end of perfusion, and if it is below 50% to administer AHG directly.

Unfortunately purified AHG is difficult to obtain and we have only been able to use it on one occasion, in the case of J.F., in whom an AHG level of 45% after perfusion was quickly restored to 60% by AHG concentrate, with cessation of bleeding.

Conclusions

It appears that a lowering of the antihæmophilic globulin level takes place while blood is slowly clotting. During perfusion on the extracorporeal circulation this takes place at a rate of about 10% every ten minutes and varies somewhat with flow rates. We feel that an AHG determination is helpful after any prolonged perfusion, and restoration to above 50% necessary to restore clotting. AHG concentrate, when available, is a better substitute than fresh whole blood or frozen plasma in infants and children.

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RÉSUMÉ

Les auteurs de cette étude se sont proposé d'évaluer certains facteurs de l'hémostase chez des malades et des animaux de laboratoire soumis à la circulation extra-corporelle. Ils ont limité leurs recherches aux variations des plaquettes, de la prothrombine, du fibrinogène et de la globuline antihémophilique au cours de la dérivation.

Le fibrinogène subit une chute de 52.8% au cours de la dérivation; on observa une diminution semblable (55.9%) dans le sang qu'on laissa coaguler dans des réceptacles siliconés. Puisque 60 mg. % de fibrinogène suffisent à combattre une afibrinémie simple, le taux moyen de 580 mg. obtenu après ces expériences semble éliminer cette cause.

Le dosage de la prothrombine indiqua tant dans le sang des sujets que dans le sang témoin une augmentation (factice) atteignant 172% après 20 minutes chez celui-là et 240% après deux minutes chez celui-ci. L'épreuve n'indique que la consommation de prothrombine sans nécessairement tenir compte de l'activation d'un précurseur. Si l'on considère une prothrombinémie à 50% ou plus comme suffisante à la coagulation, ce facteur est donc exonéré.

En dépit d'une diminution des plaquettes d'environ la moitié tant chez les animaux que chez les humains, le taux moyen après la période de circulation extra-corporelle était encore deux fois plus élevé que le niveau auquel on s'attend à l'hémorragie par thrombocytopénie. Les plaquettes ne furent donc pas en cause.

Le dosage de la globuline antihémophilique montra un abaissement proportionnel à la durée de la dérivation et au volume de sang perfusé. On nota un taux de 45 à 64% après 30 minutes et en général une diminution d'environ 10% par dix minutes. La prévention de l'hémorragie dans les blessures graves et les opérations majeures exige un niveau minimum de cette globuline de 50%. La thérapie idéale serait donc la restauration d'un taux adéquat par administration de G.A.H. Ce produit est malheureusement rare car il est d'une fabrication coûteuse. Le seul malade chez qui les auteurs ont pu l'employer pour rehausser le taux de 45% à 60% a répondu très favorablement.

SURGICAL PHILOSOPHY*

"What do we mean by surgical philosophy? The surgeon knows or should know that when he makes an incision he has damaged the body no matter how gentle and careful he has been. The area will never be the same again. No normal person wants his body marred by scars or mutilated by having parts removed. Some psychopathic individuals, on the other hand, get satisfaction by having operations performed on themselves. Surgeons must learn to estimate the realities in these cases and do their best to avoid putting one more scar on the battleground. The justification for surgery is apparent when it is the surgery of necessity, such as severe trauma, hemorrhage, perforation, obstruction, gangrene, or malignancy. It may not be quite so evident when it is the surgery of election, after medical treatment has not been successful as in duodenal ulcer, regional enteritis, ulcerative colitis, or in uncomplicated diverticulitis, hypertension, quiescent infection, or stone in biliary tract, or benign tumor, or for cosmetic purposes. The surgery of prevention or prophylaxis is another category where the justification should be under scrutiny. Some such operations are tonsillectomy, appendectomy, polypectomy, and removal of skin or breast lesions. The surgery of palliation must be carefully evaluated from the point of view of time, severity of the surgical attack, and the promise of relief. In other words, except for the surgery of necessity, the surgeon must carefully weigh the advantages against the possible results and possible complications. This amounts to the actual estimation of calculated risks. Just because a surgeon has been trained to use his techniques in an expert manner is no reason why he should make use of them indiscriminately. He should not be scalpel-happy. He should think of his patient and question whether everything has been tried that might make it possible to avoid surgery. When he is convinced and his medical confreres agree, then the prospect should be presented fairly to the patient and his relatives. It is not fair to them to ask that they make the decision. They are in no position to do this since they do not have the background to weigh the evidence. The surgeon must take the responsibility himself. The risks are his and the outcome depends upon his skill and the reaction of the patient to it. These he must be able to estimate."

*MORTON, J. J.: Surgical philosophy, *Surgery*, **44:** 927, 1958.

MEDIASTINAL AND PULMONARY ANGIOGRAPHY AS AN AID IN DETERMINING THE RESECTABILITY OF PRIMARY LUNG CANCER: A PRELIMINARY REPORT

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INTEREST IN THIS method of assessing resectability in selected cases of carcinoma of the lung has been growing since its introduction by Robb and Steinberg[8] in 1939. The procedure has been considered of value by Dotter, Steinberg and Holman,[4] Keil, Voelker and Schissel,[6] Amundsen and Sörensen[1] and, more recently, by Wyman and Wilkins.[10] These authors agree that angiography has a place in determining the extent of involvement and, therefore, the resectability in selected cases of primary pulmonary carcinoma. None are of the opinion that the procedure will establish the diagnosis of this disease.

To determine the value of the procedure, a study of resectable and non-resectable cases was initiated at the Toronto General Hospital in 1957. This report is based on the first 30 cases subjected to the examination. Only hilar lesions were chosen for this evaluation, and the accuracy of the radiological interpretation was substantiated where possible by thoracotomy.

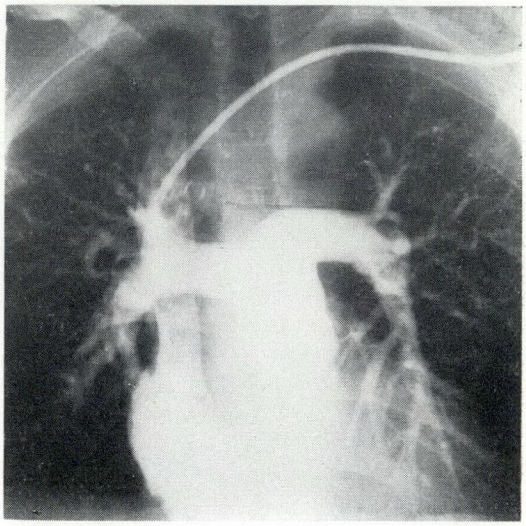

Fig. 1.—Normal frontal view. *Note:* Superior vena cava, normal right heart chambers, relative size and position of pulmonary arteries, and symmetric filling of peripheral arteries.

TECHNIQUE

All patients are examined in the supine or supine oblique position. An intravenous test dose of contrast medium is administered before beginning the procedure. After suitable premedication, the median basilic vein is isolated under local anæsthesia. An opaque Lehman catheter (size 10 or larger) is introduced and placed, under fluoroscopic control, so that its tip lies at the junction of the innominate vein and the superior vena cava. Fifty to sixty cubic centimetres of sodium acetrizoate (Urokon) (70%) is then automatically injected by a Gidlund syringe which is synchronized with an Elema biplane roll film changer. The in-

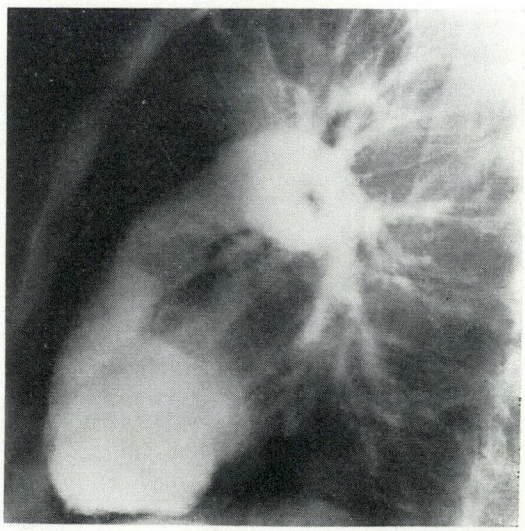

Fig. 2.—Normal lateral view, simultaneous with Fig. 1. *Note:* Position and contour of pulmonary outflow tract and superimposition of primary branches of pulmonary arteries rendering this view of limited value in detection of hilar lesions.

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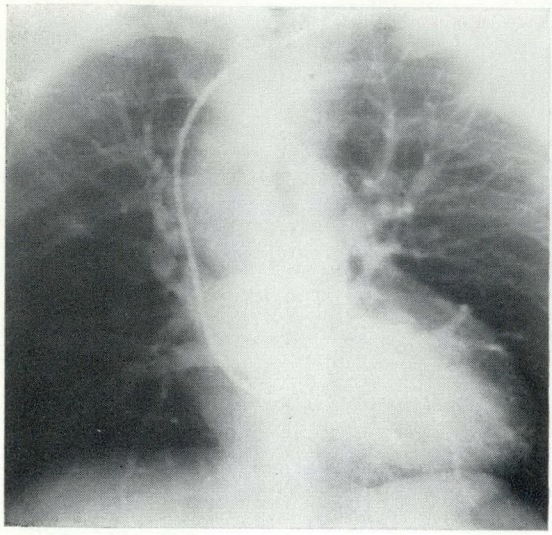


Fig. 3.

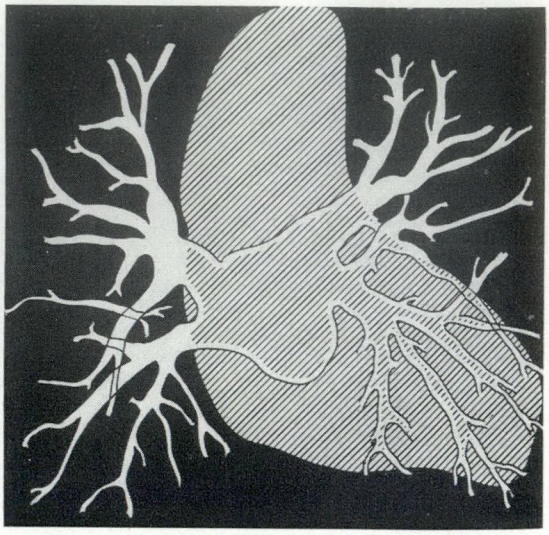
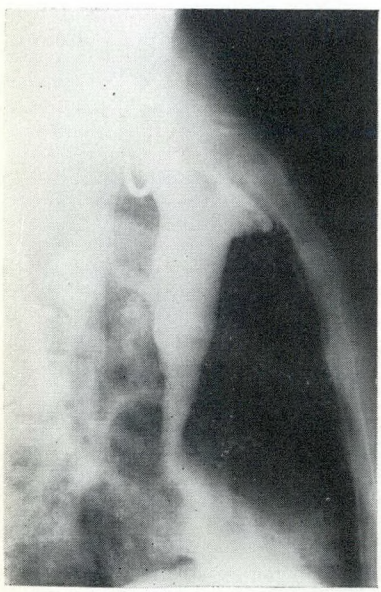


Fig. 4.

Fig. 3.—Normal pulmonary veins. *Note:* Contrast filled veins, left atrium, ventricle, and aorta. Film made eight seconds after injection. (Catheter in this case was in high right ventricle.)
Fig. 4.—Normal pulmonary veins. Direct tracing, from Fig. 3, of pulmonary venous circulation (diagrammatic).

jection phase lasts two seconds. Serial simultaneous frontal and appropriate lateral projections are obtained, initially with two exposures per second for five seconds and subsequently with one exposure per second for an additional five seconds, resulting in 15 exposures in each direction over a total time interval of 10 seconds. Stationary cross hatch grids are used. General anæsthesia is considered unnecessary. No significant reactions have been encountered in this series of cases.



F.g. 5a.

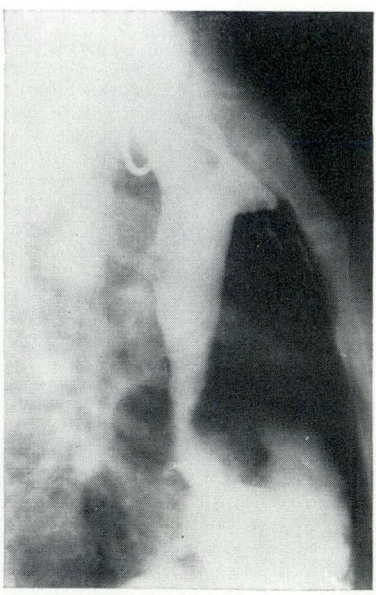


Fig. 5b.

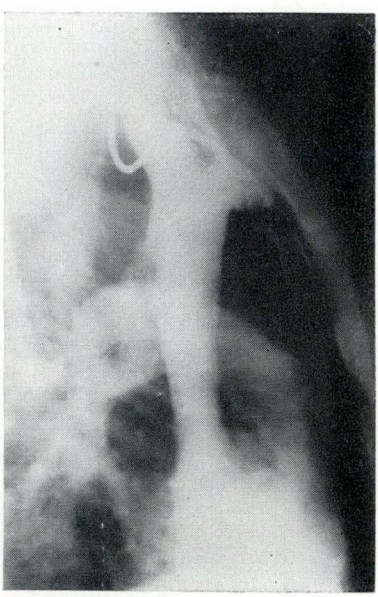


Fig. 5c.

Fig. 5a, 5b, and 5c.—Case 15. Serial lateral views showing apparent conical constriction at lower end of superior vena cava which disappears in c. This could be erroneously interpreted as a pathological defect without rapid serial films. These are made at two films per second. Superior vena cava normal at thoracotomy. Pneumonectomy.

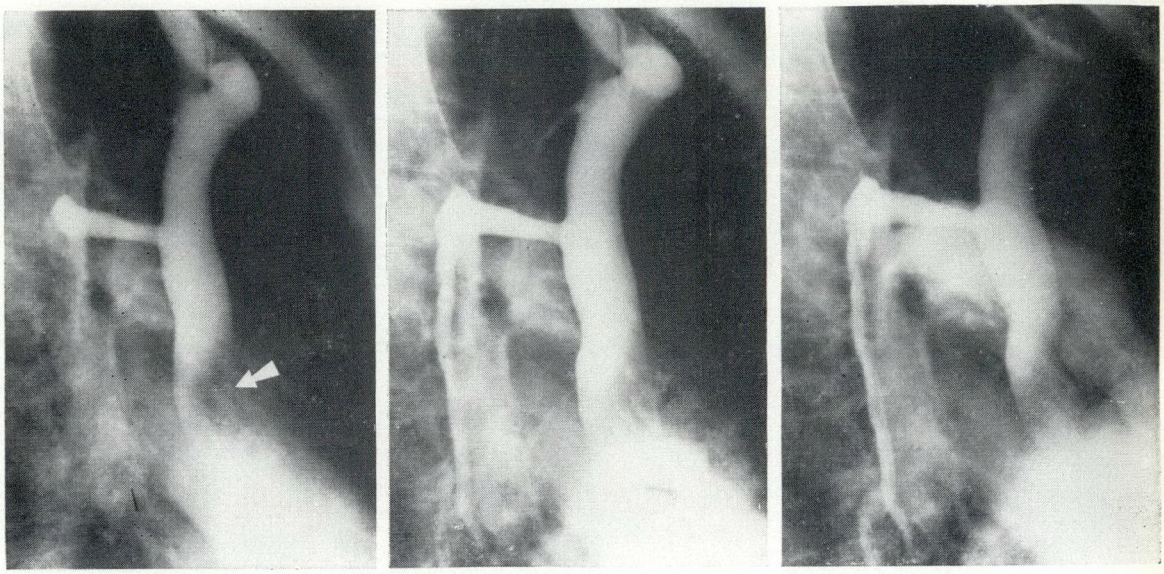


Fig. 6a. Fig. 6b. Fig. 6c.

Fig. 6a, 6b, 6c.—Case 28. Films similar to Fig. 5, but demonstrating a constant filling defect in the lower superior vena cava which is partially intra-pericardial. This indicates non-resectability. The radiological findings were confirmed at thoracotomy. *Note:* Reflux filling of azygos venous system due to partial superior vena cava obstruction which was not detected clinically.

Selection of Cases

As reported by Delarue,[3] approximately one in three of all cases of primary lung cancer subjected to thoracotomy at the Toronto General Hospital is declared non-resectable at operation. This is comparable to the findings recorded by other centres. Therefore, approximately 33% of patients undergoing surgical treatment could—and conceivably should—have been spared the distress of exploratory thoracotomy and its attendant hazards.

The criteria of non-resectability used in our cases are stringent but not dissimilar to those currently in use elsewhere. These include: obvious extension into trachea or carina on bronchoscopy, positive prescalene node biopsy, radiological evidence of extension into the chest wall, gross involvement or displacement of the œsophagus detected by radiological examination, recurrent laryngeal nerve paralysis, recovery of malignant cells from the pleural fluid, and any evidence of distant metastases. Clinical signs of superior vena caval obstruction are also considered contraindications to thoracotomy. If any of these contraindications exist, the case is considered non-resectable *a priori*. However, this report primarily deals with cases in which none of the aforementioned criteria are present. It was hoped that, by the use of angiography, another valid criterion might be added.

Significant Findings on Mediastinal and Pulmonary Angiography

Findings of significance represent evidence of involvement of the heart or great vessels by new growth. The involvement may be due to direct invasion or the result of extrinsic pressure upon these structures. The important zones under examination will now be considered separately.

1. *Superior Vena Cava and Innominate Vein*

The superior vena cava is most often involved in carcinoma arising in the right upper lobe bronchus or its branches. The physiological variation in size and contour of the great veins is marked and, for this reason, inconstant filling defects will be seen to disappear when serial films are studied (Fig. 5). The superior vena cava may show either concentric narrowing or involvement from one direction only. Obstruction of the superior vena cava below

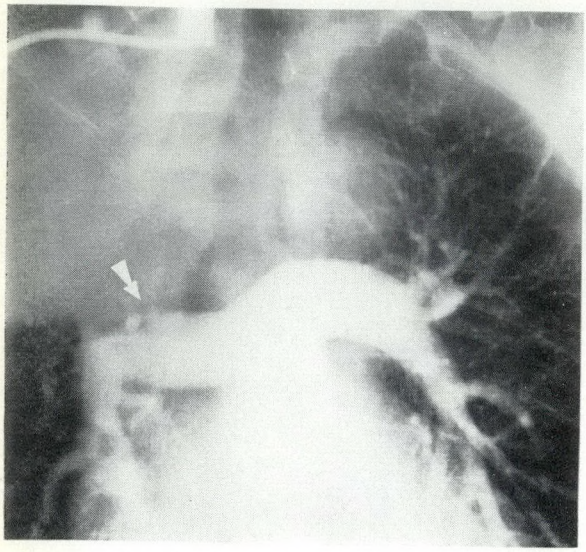


Fig. 7.—Case 1. Right upper lobe consolidation due to carcinoma arising in the right upper lobe bronchus. Complete occlusion of right upper lobe artery with filling defect in superior margin of right pulmonary artery proximal to bifurcation, indicating non-resectability.

the level of the azygos vein may be associated with reflux of contrast medium into the azygos system (Fig. 6). Influx of blood from the azygos vein will often produce a transient non-pathological filling defect.

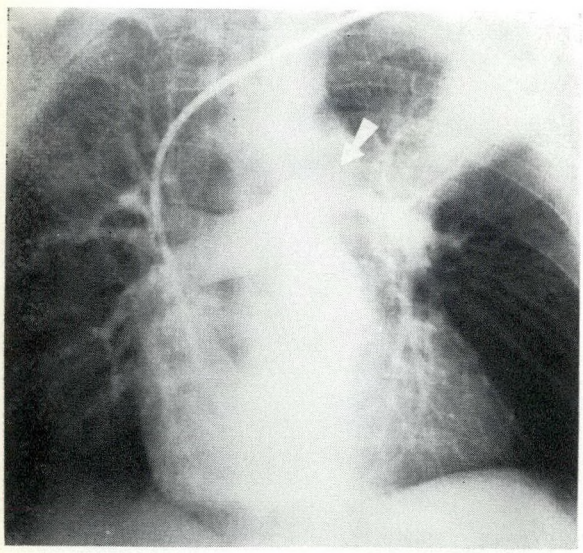


Fig. 8.—Case 4. Left pulmonary artery involved within 1 cm. of its origin. *Note:* Filling defect in supravalvular portion of main pulmonary artery and band-shaped consolidation in left upper lobe, with delay in filling of peripheral arteries on left compared to normal circulation on right.

2. Pulmonary Arterial Tree

New growth, detected by a filling defect in the main pulmonary artery, immediately rules out resection (Fig. 8). Similarly, any defect, concentric or otherwise, in the right pulmonary artery proximal to its bifurcation is indicative of non-resectability (Fig. 7). The same conclusion may be drawn when the left pulmonary artery is involved within 1.5 cm. of its origin (Figs. 8 and 10). Amundsen and Sörensen[1] have measured the height difference (H.D.) between the right and left pulmonary arteries in a similar series, and have reported that a height difference greater than 2.5 cm. or less than 1.0 cm. is suggestive of the presence of tumour in the mediastinum, causing depression or elevation of the artery on the side of the lesion. They have also stated that a left pulmonary artery diameter less than 0.8 cm. implies pathological narrowing of this vessel. They found no significant variation in the diameter of the right pulmonary artery. Our own measurements are summarized in Table I. On the basis of these findings, it is our opinion that height difference is of little practical value in detecting mediastinal spread. Indeed, our greatest height difference (3.2 cm.) occurred in a left upper lobe lesion which was later proven to be tuberculous, in which case retraction was the underlying cause of the height difference. However, the findings with regard to the right and left pulmonary artery diameters would tend to support their hypothesis.

Occlusion of the pulmonary artery branches distal to the main right or left arteries has been commonly encountered. A conical or "rat-tail" configuration is often found (Figs. 12 and 14). This lobar artery involvement does not indicate non-resectability.

Visualization of the entire length of the left pulmonary artery is rendered inadequate in the usual antero-posterior projection because it takes an antero-posterior course, and even in the lateral films inter-

TABLE I.—Average Diameters and Height Differences of Right and Left Pulmonary Arteries

Site of carcinoma	*Total cases*	*H.D. (cm.) (average)*	*Diameter RPA (cm.) (average)*	*Diameter LPA (cm.)*
Right lung.....	11	2.0 (1.4-2.5)	3.0 (2.1-3.9)	2.9 (2.5-3.7)
Left lung.....	15	1.9 (0.9-3.2)	3.0 (2.5-3.5)	2.0 (0.7-2.9)

pretation is often confused by the fact that it is superimposed on the right pulmonary artery and its branches. For this reason, we now believe that patients with left-sided lesions should be rotated into the left anterior oblique position. This provides an excellent view of the entire length of the left pulmonary artery, and the opposite oblique projection will also show the right pulmonary artery quite adequately (Fig. 12). The lateral projection, which is invariably confused by superimposition of vessels, had been of little value, apart from delineating the antero-posterior limits of the superior vena cava. In all probability, the two oblique projections will prove of greater total value, apart from this delineation of the superior vena cava.

3. *Major Pulmonary Veins and Left Atrium (Figs. 3 and 4)*

These structures are usually difficult to identify in sufficient detail to allow one to draw conclusions from the findings. Evidence of invasion of the left atrium or the pulmonary veins close to the atrium indicates non-resectability. If the veins are involved more distally, intrapericardial dissection and ligation of the veins at the atrium is feasible. Localized poor opacification of a pulmonary vein adjacent to the left atrium in the absence of arterial lesions is said to be strong evidence of neoplastic involvement.[10]

Results

The findings in the first 30 cases studied are listed in Table II. In two early cases the technique proved inadequate, and two other patients were subsequently proven to have disease other than primary carcinoma. Of the remaining 26 cases, seven were declared non-resectable on the basis of criteria listed earlier. Angiography corroborated this diagnostic appraisal in five of the seven cases.

The 19 remaining cases were considered potentially resectable before angiography. After angiography, this opinion was supported in only nine cases. In all but five instances (where operative corroboration of clinical and angiographic impressions was deemed necessary), operation was restricted to those cases declared resectable by all criteria, including angiography.

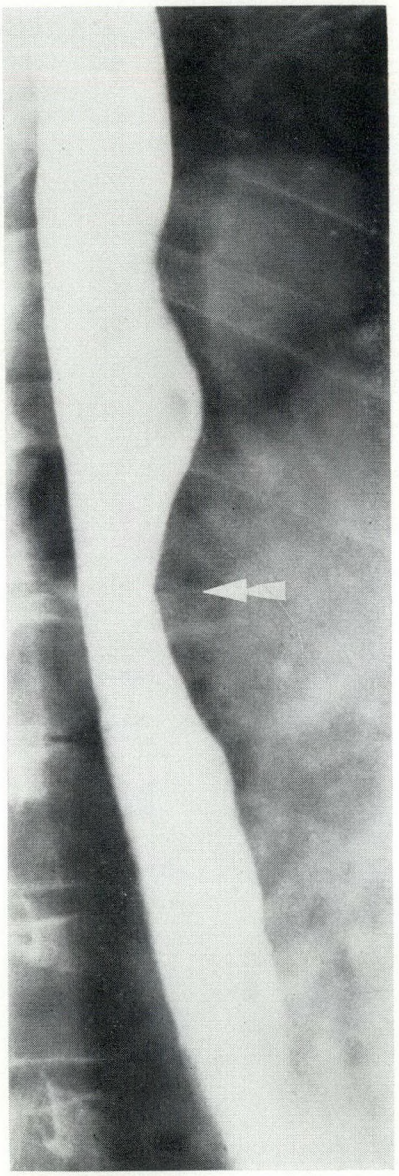


Fig. 9.—Case 21. Saucer-shaped extrinsic impression on barium-filled œsophagus due to metastases in subcarinal lymph nodes. Primary in left main bronchus non-resectable. Note normal aortic impression above.

Only one patient, predicted to have a resectable lesion by all criteria, proved subsequently to harbour a non-resectable tumour at operation. This was due to massive anterior mediastinal lymph node metastases remote from the great vessels and heart, that could not have been detected

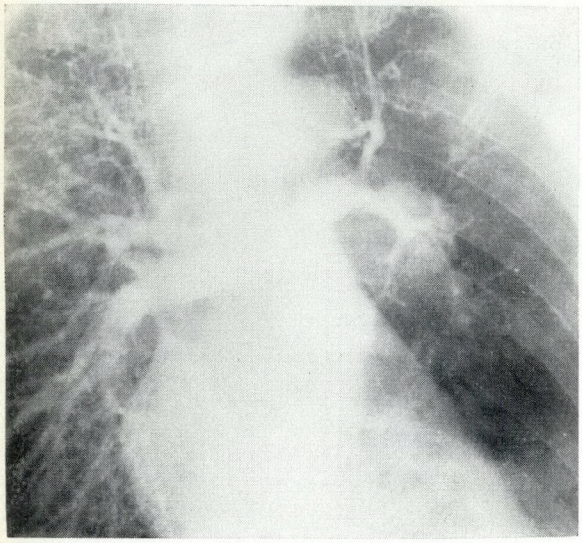


Fig. 10.—Case 6. Left pulmonary artery involved within 1.5 cm. of its origin. Almost total occlusion of left pulmonary artery with incomplete filling of peripheral branches. Non-resectable. Patient presented with central hilar mass on survey film. Asymptomatic.

by angiography in any event. The gross pathological findings in all resected specimens could be definitely correlated with the angiographic observations.

TABLE II.—MEDIASTINAL AND PULMONARY ANGIOGRAPHY A SUMMARY OF 30 CASES

Appraisal of resectability before angiography	*No. of cases*	*Appraisal of resectability after angiography*	
Non-resectable before angiography	7	1. Corroboration of non-resectability	5
		2. Resectable on basis of angiography	2
Potentially resectable before angiography	19	1. Corroboration of resectability (1 proved non-resectable at operation)	9
		2. Non-resectable on basis of angiography	10
Total cases of bronchogenic carcinoma	26		

Type of case	*No. of cases*	*Explanation*
Technically inadequate	2	1. Catheter split 2. Timing of injection and x-ray exposure not synchronized
Diagnosis other than carcinoma	2	1. Pulmonary tuberculosis 2. Pulmonary tuberculosis.
Total cases (all types)	30	

DISCUSSION

It is seen that 19 or over 50% of the cases which were considered to be otherwise resectable were declared non-resectable on the basis of angiographic findings. This is in keeping with the findings of other investigating groups.[1, 10] In our experience, height difference between the pulmonary arteries is of little prognostic value, as far

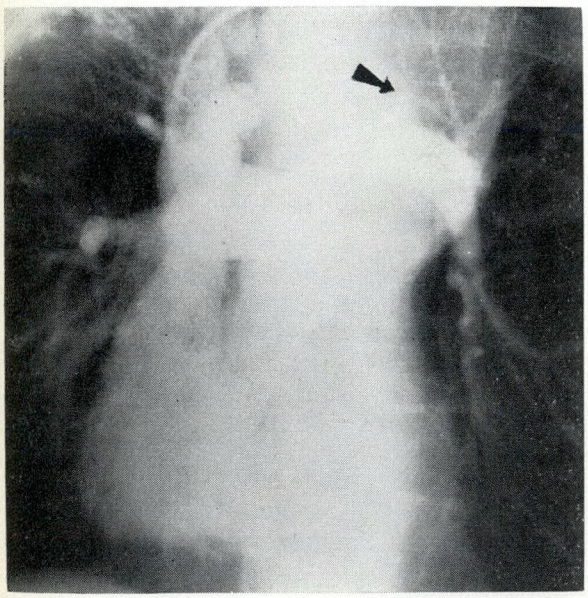


Fig. 11.—Case 19—left anterior oblique view. Shows conical occlusion of left upper lobe artery. Resectable by angiographic criteria but thoracotomy revealed involvement of left anterior vein with local spread to adjacent pericardium. Not resected.

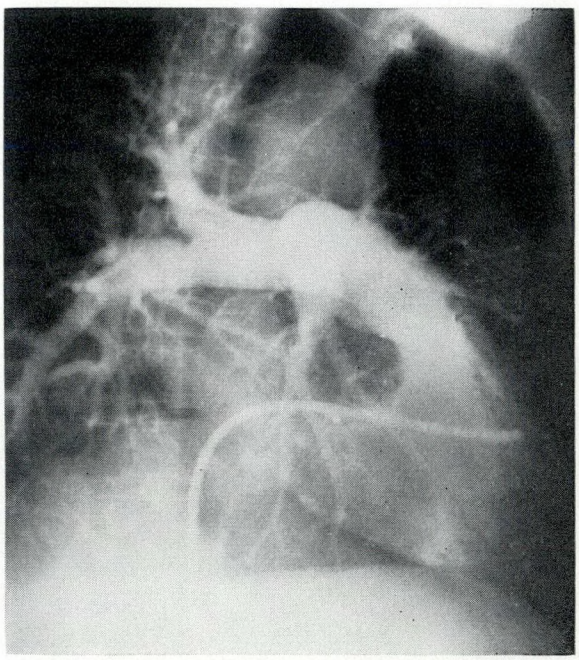


Fig. 12.—Normal case—right anterior oblique view. Demonstrating good visualization of the right pulmonary artery.

TABLE III.—Summary of 30 Cases of Primary Lung Cancer Subjected to Pulmonary Angiography at the Toronto General Hospital, 1957 - 1958

Case No.	*Pre-angiographic resectability*	*Angiographic findings*	*Predicted resectability*	*Operation performed*	*Findings at operation*
1	Resectable	RPA involved med. to bifurc.	Non-resectable	None	———
2	Resectable	LPA involved within 1.5. cm. origin	Non-resectable	None	———
3	Resectable	LPA involved within 1.5 cm. origin	Non-resectable	None	———
4	Resectable	LPA involved within 1.5 cm. origin	Non-resectable	None	———
5	Resectable	SVC and RPA involved	Non-resectable	Thoracotomy	Massive mediastinal spread
6	Resectable	LPA involved within 1.5 cm. origin	Non-resectable	None	———
7	Resectable	LPA involved within 1.5 cm. origin LUL vein occluded near atrium	Non-resectable	None	———
8	Resectable	SVC defect. RPA involved med. to bifurc. Sup. pul. vein occluded	Non-resectable	Thoracotomy	SVC and pericardium widely involved
9	Resectable	RUL artery partially occluded	Resectable	Pneumonectomy	No mediastinal spread
10	Resectable	RLL artery partially occluded	Resectable	Pneumonectomy	No mediastinal spread
11	Resectable	Normal	Resectable	Thoracotomy	Many mediastinal metastases not in association with great vessels
12	Resectable	LUL artery occluded	Resectable	Pneumonectomy	Only 2 cm. LPA to work with. Pleural spread. Not hopeful.
13	Resectable	LLL arteries partially occluded	Resectable	Pneumonectomy	Intrapericardial dissection necessary.
14	Resectable	RUL artery occluded	Resectable	Pneumonectomy	Subcarinal nodes involved.
15	Resectable	Branch of RUL artery occluded	Resectable	Pneumonectomy	No mediastinal spread.
16	Resectable	Artery to lingula occluded	Resectable	Pneumonectomy	Subpleural lymphatics involved. No mediastinal spread
17	Resectable	RLL artery occluded	Resectable	——	———
18	Resectable	RUL artery occluded; erosion 2nd rib.	Non-resectable	None	———
19	Non-resectable	LUL artery occluded	Resectable	Thoracotomy	Anterior vein and pericardium involved.
20	Non-resectable	LPA involved within 1.5 cm. origin	Non-resectable	Thoracotomy	Subpleural lymphatics and left atrium involved.
21	Non-resectable	LPA involved within 1.5 cm. origin	Non-resectable	None	———
22	Non-resectable	SVC defect. RUL artery occluded	Non-resectable	None	———
23	Non-resectable	SVC defect. LUL artery occluded	Non-resectable	None	———
24	Non-resectable	Normal	Resectable	None	———
25	Non-resectable	SVC constricted. RUL artery occluded	Non-resectable	None	———
26	———	Technically inadequate	———	———	———
27	———	Technically inadequate	———	———	———
28	Tuberculosis	Normal	———	———	———
29	Tuberculosis	Anomalous branching RUL artery	———	———	———
30	Resectable	LPA involved within 1.5 cm.	Non-resectable	Thoracotomy	LPA involved. Non-resectable.

as resectability is concerned. It is probably correct to say that a left pulmonary artery diameter of less than 0.8 cm. is indicative of pathological involvement and non-resectability. No significance can be attached to minor degrees of narrowing when they are uniform and unassociated with a localized pressure defect, in view of the wide normal variations found here. The diameter of the right pulmonary artery showed no correlation with the incidence of its pathological involvement.

It is therefore suggested that this method of examination may, potentially, reduce by one-half the number of patients subjected to a fruitless thoracotomy. The difference in the number of patients pronounced non-resectable after angiography (50% approximately) and the number previously considered non-resectable after thoracotomy (33%) is sizable. This is, in all probability, accounted for by two factors. In the first instance, some palliative resections are carried out with beneficial symptomatic effect but not with any intention of eradicating the disease, and in the second place the present series of angiographic studies affects such a small number of cases that it cannot be considered statistically significant. Nonetheless, the five instances in which obvious clinical non-resectability was corroborated by angi-

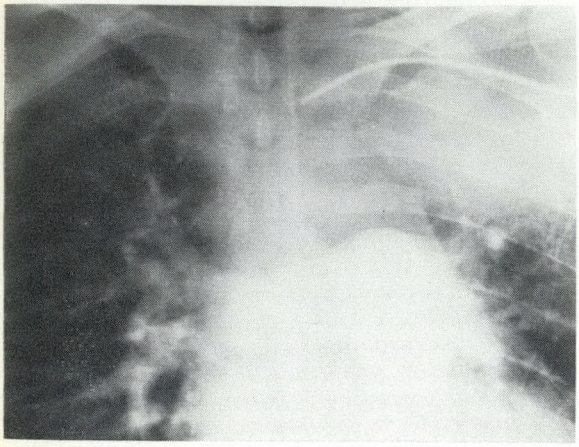


Fig. 13.—Case 20. Left pulmonary artery involved within 1.5 cm. of its origin. Non-resectable. Thoracotomy proved non-resectability with involvement of left atrium and subpleural lymphatics as well. Biopsy—alveolar cell carcinoma. This is the only case included which did not have a predominantly hilar lesion.

ography in a group of seven patients of this type implies that this radiological procedure is potentially accurate in delineating the anatomical extent of the mediastinal extension.

In all probability angiography will, in the future, be indicated only in centrally situated bronchogenic carcinomata considered resectable by all other methods of investigation. The cost of the examination in time and materials is prohibitive and for this reason too it should not be undertaken indiscriminately.

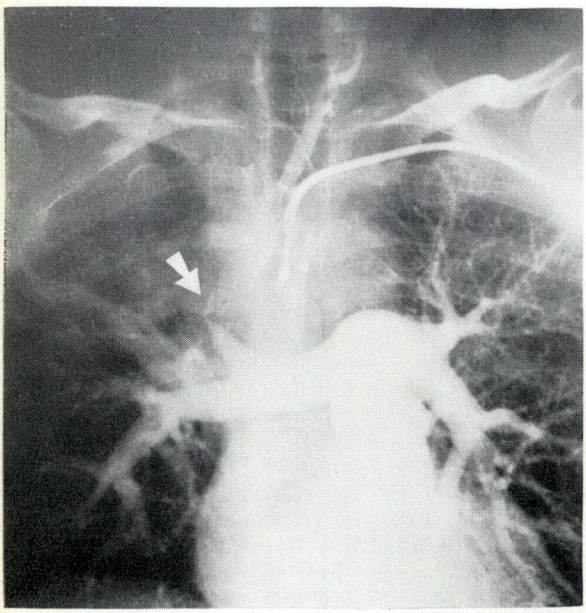


Fig. 14.—Case 14. *Note:* Normal right pulmonary artery but conical occlusion of right upper lobe artery, and wedge-shaped atelectasis in right upper lobe. Pneumonectomy carried out. Subcarinal nodes resected showed metastatic involvement.

Summary

The aims and technique of mediastinal and pulmonary angiography are outlined, insofar as they affect the diagnostic appraisal of the anatomical extent of bronchogenic carcinoma.

Angiographic findings indicative of non-resectability are discussed.

The findings in 30 cases are presented. Nine of the 19 cases, previously considered resectable, were declared non-resectable on the basis of this procedure. In addition, non-resectability was corroborated in five of seven cases with other evidence of non-resectability.

It seems likely that the use of angiography will be restricted to those cases considered resectable by all other criteria save thoracotomy.

Acknowledgments

The authors would like to express their sincere gratitude to the Ontario Cancer Treatment and Research Foundation which supplied the funds necessary for this investigation. Without this financial aid, the program could not have been carried out.

The advice of colleagues Dr. R. B. Holmes and Dr. F. C. Parrott is also gratefully acknowledged.

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RÉSUMÉ

L'angiographie médiastinale et pulmonaire permet d'estimer l'opérabilité des cas de carcinomes pulmonaires. Cette méthode prend de plus en plus d'importance. Dans le but de préciser sa valeur, les auteurs l'ont essayée dans une série de 30 cas de cancers du poumon considérés cliniquement comme opérables ou non. On a choisi uniquement des lésions du hile et les interprétations radiologiques furent controlées par la thoracotomie toutes les fois que la chose fut possible.

Technique.—Sous anesthésie locale, on dénude la veine médiane basilique et l'on y introduit un cathéter opaque aux rayons-x (cathéter de Lehman); celui-ci est poussé, sous contrôle radioscopique, jusqu'à la confluence de la veine innominée et de la veine cave supérieure. On injecte alors 50 à 60 c.c. d'acétrizoate sodique (Urokon) à 70% en deux secondes. Des clichés sériés sont pris dans les plans frontaux et latéraux, à raison de 15 en dix secondes. Il n'a jamais été observé de complications sérieuses. Les radiographies obtenues traduisent le degré d'envahissement du cœur et/ou des gros vaisseaux par le processus tumoral.

La veine cave supérieure est très souvent touchée dans les cancers de la bronche supérieure droite. Un défaut de remplissage de l'artère pulmonaire souche ou de ses deux grosses branches contre-indique radicalement la résection. De même pour ce qui est de l'envahissement des veines pulmonaires à proximité de leur abouchement dans l'oreillette. D'une façon générale, les images pathologiques obtenues sont des rétrécissements du calibre du vaisseau, concentriques ou non. Il a été possible d'établir des normes des diamètres vasculaires.

Avec cette méthode on a pu établir qu'en moyenne 50% des cas considérés comme cliniquement opérables, se sont révélés être en réalité inopérables. Ceci permettra d'éviter aux malades les hasards d'une thoracotomie exploratrice inutile.

THE CASE FOR CONSERVATISM IN CANCER*

"There are at least three biological types of cancer: (1) cancers that do not tend to metastasize, (2) those that metastasize chiefly to the regional lymph nodes, and (3) those that, even before the primary tumor is recognized, have metastasized systemically. Radical operations are not necessary in the localized group and do not cure the patients with cancers that have already spread through the blood stream. It is only in treatment of cancers that tend to metastasize solely to regional lymph nodes that radical resections of lymph nodes are of value.

"In some kinds of cancer, like squamous cell cancer of the mouth, metastasis limited to the regional nodes is so common that radical operations are well justified. In other kinds of cancer, like those of the lung, the tumors tend to be so widely disseminated before the presence of the primary tumor is recognized that little is gained by extending the scope of the operation. In these cases, involvement of lymph nodes is more a sign of incurability than an indication for extending the scope of the operation. Finally, there are many cancers, like cancers in a polyp of the rectum, which tend to be localized and can be cured by local treatment. If extensive operations are done to cure such localized tumors, the morbidity and mortality of the operations far exceed the possible dangers of recurrence after local treatment.

"Overtreatment of cancer may be just as dangerous as undertreatment. A proper balance is a matter of clinical judgment in each individual case. No man-made principle of cancer surgery can be applied successfully to the treatment of all cancers."

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*CRILE, G., JR.: The case for conservatism in operations for cancer, S. *Clin. North America*, **38:** 1215, 1958.

PROGNOSIS IN LUNG CANCER SURGERY BASED ON BLOOD VESSEL INVASION*

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IN THE NINE YEAR period from January 1, 1949 to December 31, 1957, 329 cases of lung cancer were diagnosed at the Chest Clinic of Notre-Dame Hospital, Montreal. In 75 of these cases, the diagnosis was of a clinical nature only, leaving 254 cases histologically proven by bronchoscopic examination, biopsy of lymph nodes, thoracotomy or autopsy. A follow-up was obtained of all cases in the histologically proven group. The age and sex incidence was the usual encountered in this disease.

TABLE I.—RESECTION OF LUNG CARCINOMA, 37 CASES, NOTRE-DAME HOSPITAL

Resections	37
Died of secondaries	19*
Died of other causes	6
Living with secondaries	3
Living and well *more* than 2 years	7†
Living and well less than 2 years	2

*Average survival 8.2 months (min. 3, max. 22).
†2 to 8 years.

In only 77 (30%) of the proven cases was a surgical attempt at cure possible. Out of the 77 patients, only 48 could undergo resection, 11 of whom died within 30 days of surgery, leaving a total of 37 operative survivors whose follow-up over the period mentioned can be considered. Before May 1953, a so-called "standard" resection leaving the majority of the mediastinal lymph nodes was carried out. After this date, a more radical approach to pneumonectomy was taken without increasing the mortality rate, but it remains to be seen whether the more radical dissection of the upper and lower mediastinum carried out in this fashion is worth while.

Table I shows that of the the 37 patients who underwent resection, 19 (or 51.3%) died of secondaries with an average survival time of 8.2 months. Four patients only survived more than one year, with a maximum survival in one patient of 22 months. As it was noted that the major cause of death in this group was blood-borne metastases, a histological review of these specimens was carried out by one of us (C.G.-M.). This review was made in order to study blood vessel invasion in the lung. No previous knowledge of the clinical course in each case was available to the pathologist during the examination of these slides.

One specimen was excluded from the study because the lung had been kept as a museum specimen. This was an alveolar cell carcinoma in a woman, and the patient is living but with secondaries four and a half years after resection.

In Table II, the clinical course is correlated with vascular and lymphatic invasion. It appears evident from this that, in this small series, all the patients who survived more than two years or who died of other causes had neither blood vessel nor lymph node invasion. In contrast to this, all the patients living with secondaries or living and "well" less than two years have involvement either of blood vessels alone or of blood vessels and lymph nodes, and 94.7% of patients who died of secon-

TABLE II.—CORRELATION BETWEEN CLINICAL COURSE AND VASCULAR AND LYMPHATIC INVASION

Resected cases	*No. of cases*	*V.I.*	*L.I.*	*V.I. and L.I.*	*Negative*	*% of V.I.*
Living and well, more than 2 years	7	0	0	0	7	0%
Living and well, less than 2 years	2	1	0	1	0	100%
Living with secondaries	2	1	0	1	0	100%
Died of secondaries	19	9	1	9	0	94.7%
Died of other causes	6	0	0	0	6	0%
	36	11	1	11	13	61.1%

V.I.—Vascular invasion.
L.I.—Lymphatic invasion.

*From the Departments of Surgery and Pathology, Notre-Dame Hospital, Montreal.

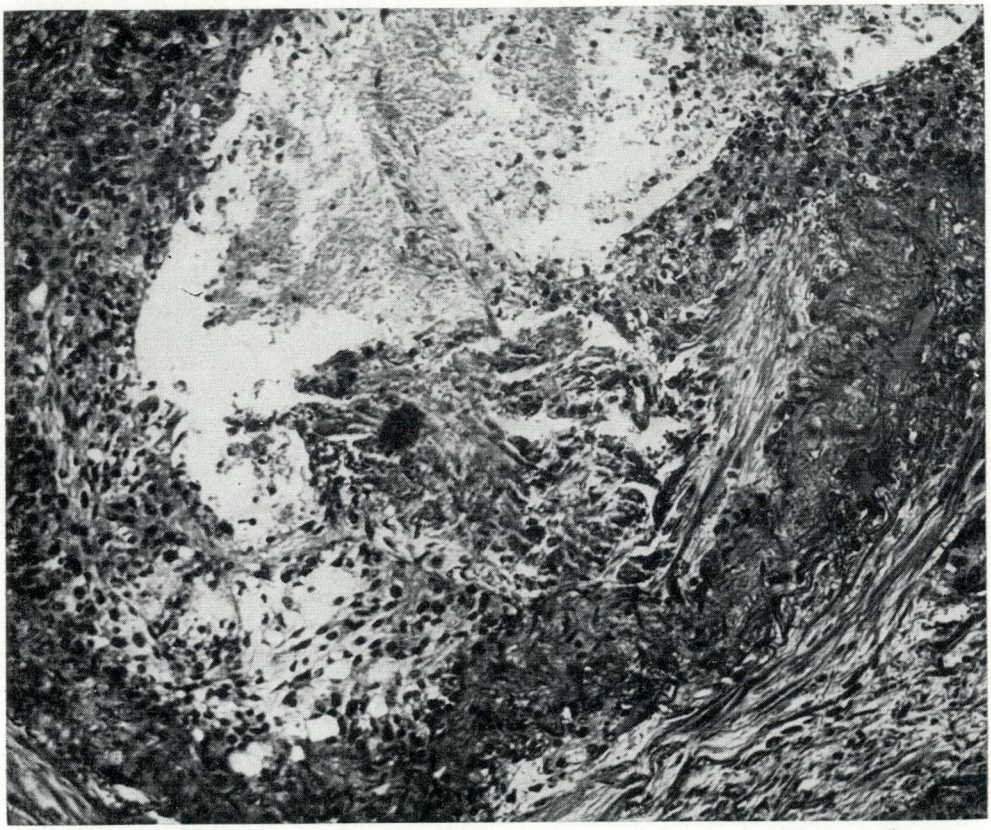

Fig. 1.—(Medium magnification). This shows the wall of a vessel in the right lower corner. It is invaded by tumour cells which are seen proliferating inside the lumen in the centre. The tumour cells are in contact with the blood stream. This patient died from cerebral metastasis a few months after operation.

daries had vascular invasion. In one case only was there lymph node invasion alone.

These figures, admittedly in a small series, appear significant and logically are quite understandable if one remembers that the lung is a vascular sponge. Obviously the presence of cancer cells in the smaller vessels will influence the survival rate in resected cases. The correlation between vascular invasion in the specimen and the clinical course is illustrated by the accompanying photomicrographs with a summary of the clinical course in each case (Figs. 1 to 4).

Comments

For each of the 36 cases, from five to 10 slides were examined carefully. The photomicrographic pictures illustrated in Figs. 1 to 4 are typical examples and were encountered repeatedly on the same slides. As pointed out by others,[1] no distinction between vein and artery could be made because of the smallness of the vessels.

It is interesting to note in Fig. 2 that the tumour cells from a very necrotic tumour seem to remain viable in the blood stream; this probably explains the death from blood borne secondaries in this patient even though the lymph nodes were negative.

The intimal proliferation noted in Fig. 3 was found in the majority of the slides examined from the long term survivors. Whether this intimal reaction is initiated by the tumour itself, or is a peculiar reaction of the host to his cancer, is unknown. Nevertheless it appears to be an adequate defence (for a time) against metastases. This seems to be borne out in Fig. 4, where tumour cells are invading newly formed channels in the obliterated lumen. This

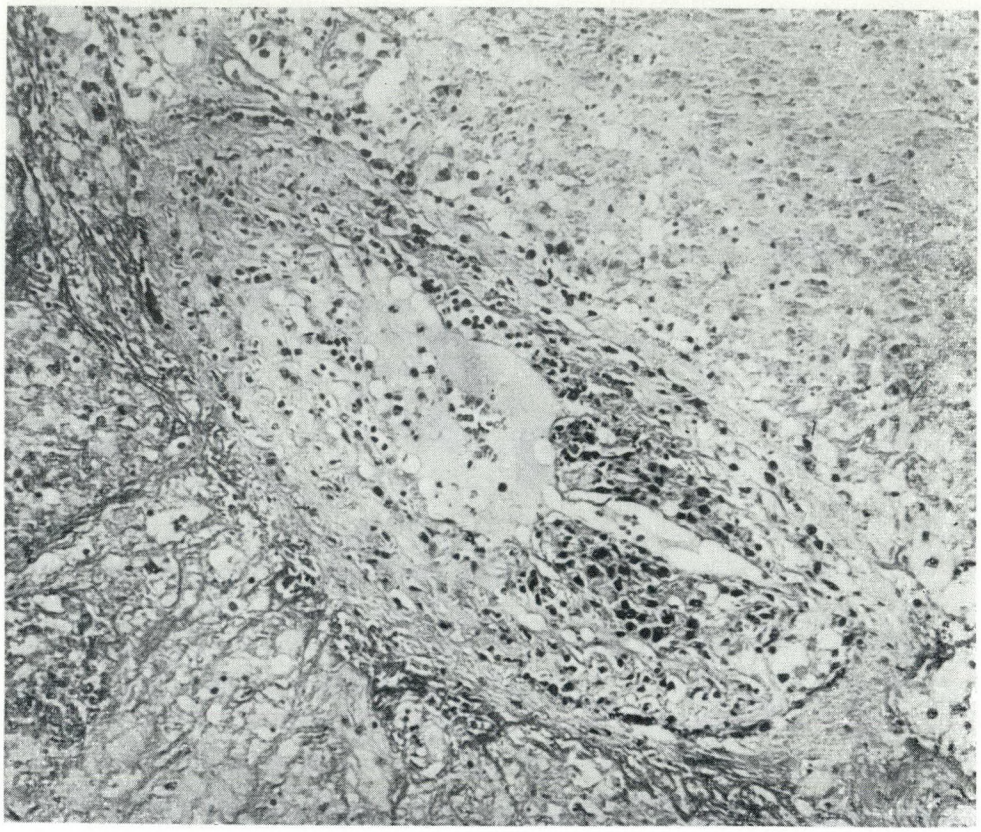

Fig. 2.—(Medium magnification). This is a largely necrotic tumour as can be seen in the right upper corner and left lower corner. In the lumen of the vessel, in the centre, there are viable tumour cells that seem to remain alive only because of their position. The patient died with blood borne metastases six months after resection. The lymph nodes were negative.

patient had a circumscribed lesion in an upper lobe for which he refused operation for 11 months. He finally agreed and resection was performed, but he died of brain secondaries 14 months later. His proliferative reaction might have enabled him to survive, had he agreed to operation sooner.

It is noteworthy that in this small series the critical survival period appeared to be two years. No patient who died of secondaries survived beyond 22 months. This is in contrast to certain larger series, from the United States or elsewhere, where it is said that one may expect a small percentage of cures even with positive mediastinal lymph nodes, though this is exceptional. Other authors[2] have reported a 6% five year survival in patients with blood vessel invasion, and 75% survival in those without. Their series being based on a much larger number, it is likely that our figures, when enlarged, will compare favourably. So far in our experience all patients with blood vessel invasion have died within two years, and survival beyond this period can be expected only in those patients who have neither blood vessel nor lymphatic invasion. It would therefore seem important to carry out a more careful histological examination of the specimen after operation, with particular attention to vascular invasion. In this way, the patient, his family and his doctor can be informed of the possible prognosis in each individual case.

The histological type in our experience seldom bears any direct relation to the prognosis except that the epidermoid type, growing more slowly, may possibly produce more intimal proliferation and therefore less vascular invasion. Furthermore,

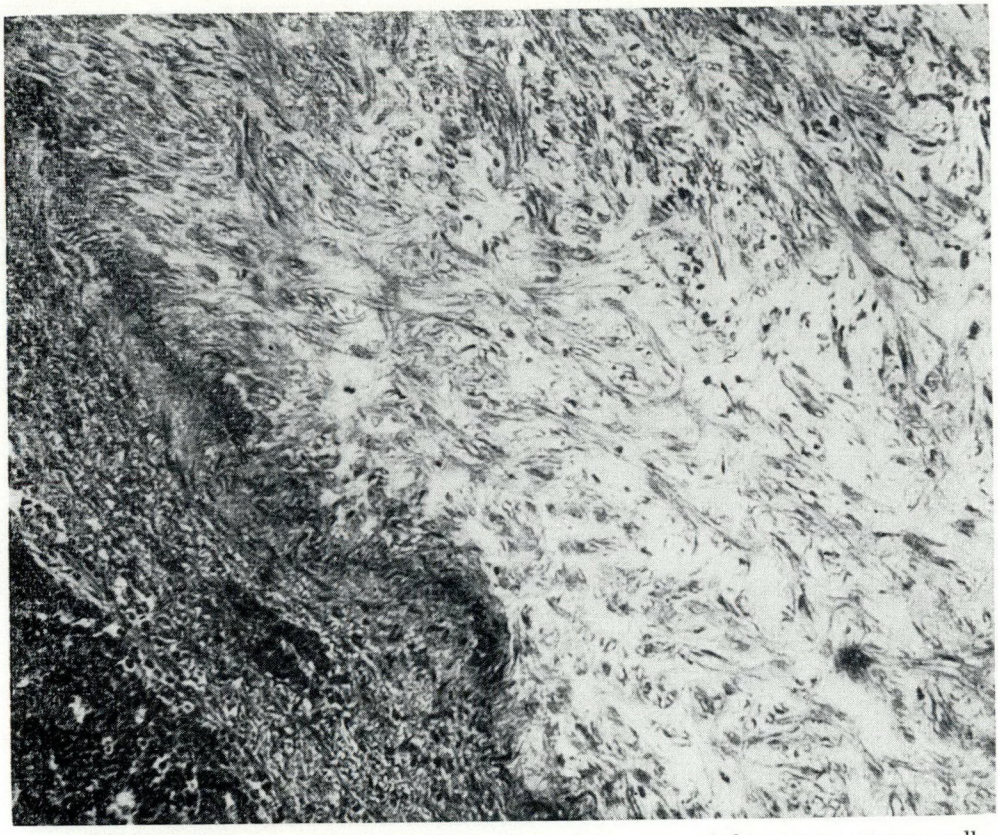

Fig. 3.—(Medium magnification). In the lower left corner of this picture, tumour cells can be seen growing up to the vessel wall. The lumen of this vessel is obliterated by a proliferation of the intima and we can see the newly formed vascular channels within the obliterating sclerosis. The patient is well three years after resection.

we thoroughly agree with Wartman[3] that in bronchogenic carcinoma "examination of large sections or enough small sections will reveal varied histologic patterns." In this way, our cases are classified as predominantly epidermoid, predominantly "oat-cell", etc.

The importance of early diagnosis and operation as the only possible chance of cure is the conclusion to be drawn from Table II which shows that 61.1% of 36 cases had vascular invasion at operation. Only 13 or 36% had neither vascular nor lymphatic invasion and could hope for a chance of cure.

Conclusions

Thirty-seven survivors of lung resection for carcinoma were studied with special attention to invasion of blood vessels in the surgical specimens.

It was noted that all patients surviving and "well" more than two years had no blood vessel invasion in the removed lung.

All patients dying of other causes had no blood vessel invasion.

All but one of the patients dying from secondaries had blood vessel invasion.

The obliteration of lung vessels by proliferation of the intima seems to be an effective barrier to secondaries.

The histological type of the carcinoma does not seem to influence the prognosis directly.

The presence or absence of blood vessel invasion, more than any other criterion, seems to us important in the prognosis of lung cancer. More attention should be paid to this as an aid to prognosis. Operation at the earliest possible time affords the only chance of cure.

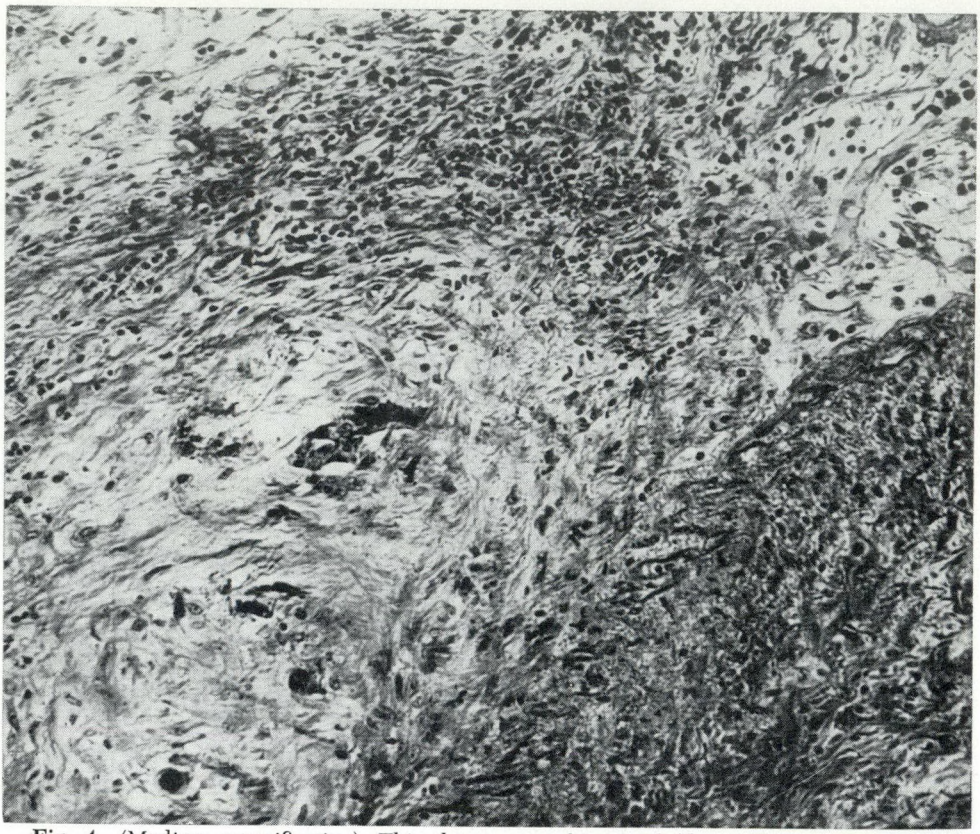

Fig. 4.—(Medium magnification). This shows part of a vessel (the wall can be recognized in the lower right corner) that has been completely obliterated by intimal proliferation, but we can clearly see several tumour cells in the newly formed channels in the centre. This patient refused operation for 11 months and died from cerebral secondaries 14 months after operation.

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Résumé

Nous avons fait une étude histologique attentive de 37 poumons prélevés chirurgicalement à l'hôpital Notre-Dame depuis neuf ans. Sur 329 cas diagnostiqués, 254 ont été prouvés histologiquement dont 77 seulement semblaient opérables. De ces derniers, 48 ont subi des résections parmi lesquels 11 sont morts dans la période post-opératoire.

L'étude a donc été faite sur les 37 cas restants; nous avons entrepris ce travail pour rechercher l'envahissement vasculaire par les épithéliomas du poumon sur les pièces chirurgicales parce que nous avons remarqué que la plupart des patients qui mouraient dans les deux ans après l'intervention avaient fait des métastases par voie sanguine, souvent sans envahissement des ganglions du médiastin, à l'opération.

Les chiffres obtenus en compilant les résultats sont assez frappants comme le montre le Tableau II. En effet, (1) tous les patients qui survivent "bien" plus de deux ans n'avaient pas d'invasion des vaisseaux sanguins dans le poumon enlevé; (2) tous les patients qui sont morts d'autres causes que de métastases n'avaient pas d'envahissement vasculaire; (3) tous les patients morts de métastases, sauf un, avaient des envahissement vasculaires.

De plus, il semble que l'oblitération des vaisseaux par la prolifération de l'intima, rencontrée fréquemment dans les cas sans envahissement, soit un mécanisme de défense qui agit au moins un certain temps pour retarder l'essaimage de l'épithélioma.

Cependant, nous ne croyons pas que le type histologique nous permette de porter un pronostic valable parce que l'apparence histologique d'une tumeur varie souvent selon les différentes régions de la même tumeur.

En somme, même avec cette petite série de cas, il nous semble possible de montrer que l'absence ou la présence d'invasion vasculaire est le critère le plus important si on veut essayer de faire un pronostic dans chaque cas et qu'il reste toujours vrai que l'opération précoce offre la seule chance de guérison.

THE COMPLICATIONS OF PULMONARY RESECTION IN TUBERCULOSIS*

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FOR ALMOST A CENTURY collapse procedures, primarily thoracoplasty, have been the mainstay in the surgical management of pulmonary tuberculosis. With the advent of specific chemotherapy, however, resection of the diseased portions of lung has become safer and is now considered the preferable approach to selected lesions. Unfortunately the pendulum has swung too far in many centres where thoracoplasty has been abandoned and where, with misplaced pride, surgeons state that they have not performed a primary thoracoplasty "for years". The bronchopleural fistula rate alone in over 7000 resections reported from the current literature is 6.7%.[1] This implies that resections are being carried out where a thoracoplasty would have been wiser. The overall morbidity rate in pulmonary resection in tuberculosis lies between 15 and 30%. For these reasons and also because we have comparable groups of white patients and North American Indians we felt that the complications encountered in 150 consecutive resections and their management could be usefully recorded.

CLINICAL MATERIAL

One hundred and fifty consecutive resections performed on 148 patients form the basis of this report. Eighty patients were North American Indians (mostly Crees)—35 males and 45 females. Sixty-eight patients were whites—30 males and 38 females. The age distribution of the whole group is shown in Fig. 1.

Of the 148 patients 139 had a positive sputum upon admission to sanatorium and 46 were not only still sputum positive but also drug resistant (to more than one of the three anti-tuberculous drugs – para-

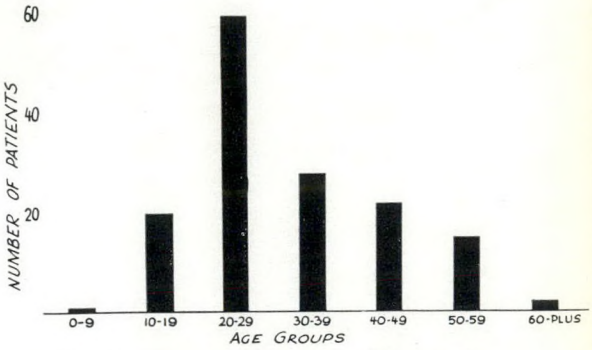

Fig. 1.—The age incidence in 150 resections.

aminosalicylic acid (P.A.S.), isoniazid, or streptomycin) at the time of operation. The reasons for resection were predominantly presence of residual disease and destroyed lung tissue (Table I). Three patients were operated upon for bronchopleural fistulæ after operation elsewhere, and further resections were performed and the fistulæ closed.

Preoperative bronchoscopy was routine to exclude patients with active endobronchitis, and bronchography was done in most patients to locate the diseased areas so that a minimum of lung tissue could be resected. Pulmonary function studies were done on all salvage cases and on many of those over the age of 40. Associated diseases were controlled before operation. Although we believe in comprehensive physiotherapy preoperatively and postoperatively, we have no physiotherapists at the Saskatoon Sanatorium, but all patients are taught diaphragm control, deep breathing exercises and coughing techniques by the nurses.

Although the incidence of pulmonary tuberculosis is higher among the Indian population, the surgical procedures neces-

TABLE I.—INDICATIONS FOR RESECTION

Residual cavities	41
Residual caseonodular lesions	38
Destroyed lobes	36
Destroyed lungs	24
Tuberculomata	8
Bronchopleural fistulæ	3

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TABLE II. — Surgical Procedures in White and Indian Patients

Procedures	*Numbers*	*Whites*	*Indians*	*Per cent*
Segmental resection:				
Alone	44	23	21	33.3
With thoracoplasty	6	3	3	
Lobectomy:				
Alone	51	19	32	
With thoracoplasty	13	4	9	48.7
With segmental resection	9	4	5	
Pneumonectomy	26	14	12	17.3
Wedge resection	1	1	—	0.7

sary for white and Indian patients differ little (Table II). Lobectomy accounts for almost half of the resections whereas wedge resection is rarely practicable in our hands.

Complications of Resection and Their Management

In this group of consecutive resections 36 patients (24%) developed 40 complications and five died within 60 days of surgery (Table III). There was a slight difference between the races in that 21 whites (31%) and only 15 Indians (19%) developed complications. The highest incidence (18 complications) was in the 20 to 29 year age group, followed by the 30 to 39 year age group (10 complications), but these were the decades containing more than half the patients and in which most of the segmental resections were done. Drug resistance and a persistently positive sputum were present in all cases with serious complications but the type of operation, i.e. segmental resection, more than anything else was the determining factor in the incidence of complications. Neither age, sex nor race seemed to influence the complication rate in contrast to other series.[2] Of the 36 patients developing complications seven were sputum positive and drug resistant at the time of operation.

Deaths.—Five patients died after operation. One patient died from a pulmonary embolus five weeks after right upper lobectomy. One patient died 12 hours after operation from acute pulmonary œdema complicating a transfusion reaction. Two patients after salvage resections died of respiratory insufficiency on the fifth postoperative day – one after pneumonectomy and one after multiple segmental resection and decortication. A further patient with salvage resection died from secondary hæmorrhage five weeks after right upper lobectomy combined with an apical lower segmental resection, and two weeks after a Schede thoracoplasty for bronchopleural fistula complicating the first operation. Only one death, due to a transfusion reaction, occurred in an Indian patient.

Bronchopleural fistula. – Two patients developed bronchopleural fistulæ. One followed a staphylococcal empyema complicating an apico-posterior segmental resection in a young man. This responded to tube drainage and a five-rib standard thoracoplasty. The second fistula occurred after a salvage resection in a middle-aged man. A right upper lobectomy and apical lower segmental resection were done in conjunction with a five-rib thoracoplasty and a tracheotomy. The right upper lobe bronchus broke down. A Schede thoracoplasty was performed but the patient died two weeks later from secondary hæmorrhage (vide supra).

Bronchopleural fistula is better prevented than cured, and careful attention to bronchus closure will minimize its occurrence. A short vascular stump, well covered and

TABLE III.—Complications of Resection

Procedure	*No. of patients*	*Post-op. hæmorrhage*	*Paralytic ileus*	*Air leak*	*Atelectasis*	*Fibrothorax*	*Wound infection*	*Residual pleural space*	*Empyema*	*Broncho-pleural fistula*	*Pulmonary embolism*	*Pulmonary insufficiency*	*Transfusion reaction*
Segmental resection	17	—	—	13	—	1	—	2	1	1	—	1*	—
Lobectomy	14	1	1	2	2	1	1	4	—	1*	1*	—	1*
Pneumonectomy	5	2	—	—	1	—	1	—	1	—	—	1*	—
Total	36	3	1	15	3	2	2	6	2	2	1	2	1

*These patients died from their complications.

closed with wire is essential for bronchus closure. In spite of such principles the incidence of bronchopleural fistula remains around 7% after pulmonary resection for tuberculosis. Once a fistula develops the mortality rate is around 25%.[3] An attempt should be made to close the stump again and a thoracoplasty should be done, or a pre-existing thoracoplasty converted to one of the Schede type. By these means it is possible to salvage about half of the cases.

Empyema. — Two patients developed empyema. In one patient (vide supra) a staphylococcal empyema complicated a segmental resection. The infection and bronchopleural fistula both responded to tube drainage and thoracoplasty. The second, a case of salvage resection, developed a tuberculous empyema after left pleuropneumonectomy for a destroyed lung. This responded to an eight-rib thoracoplasty five weeks after the original resection.

Empyema may occur alone or may complicate or produce a bronchopleural fistula. When it develops, drainage of the infected space, closure of the fistula if present, massive chemotherapy and thoracoplasty are the measures to adopt. These will succeed in most cases except when a bronchopleural fistula exists. Again prevention of empyema by careful bronchus closure, by extrapleural mobilization of cavitary lesions and by pleural toilet, particularly against spillage, will decrease the danger of infection of the pleural space.

Air leaks and persistent pleural space.— These two conditions account for more than half of all the complications of pulmonary resection. They followed segmental resection, or lobectomy when an incomplete fissure separated the lobes — almost always right upper and middle lobes—in 21 patients.

Air leaks may persist from the time of operation or may suddenly develop five to fifteen days afterwards. The management is simple. The tube is promptly reinserted or the existing tube is left in and suction applied until the leak is minimal or ceases. This may be for from two days to two weeks, and when the tube is removed a persistent pleural space often remains. This is no cause for alarm[4] and thoracoplasty has never been necessary in our experience. With present-day chemotherapy and with posture, these spaces gradually close without complication. The incidence of air leaks and persistent pleural spaces is said to be reduced by a simultaneous space-reducing thoracoplasty. We have used this in only 20 resections. We are not convinced that such space-reducing operations should be routine; they should be used only when significant disproportion between the residual pleural space and the remaining lung will exist if the chest cage is not reduced in size.

Atelectasis. — Three patients developed atelectasis. Two of these were controlled by bronchoscopic aspiration while the third needed a tracheotomy to maintain a clear airway. Atelectasis should be rare if comprehensive physiotherapy is continued in the postoperative period. Careful tracheal toilet during operation and careful aspiration of the airway at the end of the operation by the anæsthetist also help to minimize postoperative lung collapse. Should it develop, vigorous physiotherapy, bronchoscopy and tracheotomy, if more conservative measures fail, are necessary to encourage re-expansion.

Hæmorrhage.—Three patients had serious postoperative bleeding. Two of these episodes complicated difficult pleuropneumonectomies and responded to transfusions and aspiration of the pleural space. The third was the result of a ligature slipping off the posterior segmental artery 24 hours after a right upper lobectomy. The chest was promptly reopened and the vessel religated. Most postoperative bleeding is the result of extensive stripping of adherent lungs from the chest wall, and careful note of the postoperative chest drainage is necessary. Such bleeding usually responds to conservative measures but the rare exception must be recognized.

Miscellaneous complications.— Paralytic ileus has occurred in one elderly Indian woman and responded to intestinal decompression and parenteral fluids. Some degree of fibrothorax resulted after two difficult lobectomies, and two patients had significant wound infections which responded to drainage and antibiotics.

TABLE IV.—Ventilation and Blood Gas Determinations in 16 Cases of Salvage Resection

Patient			Operation	% of Pred. M.B.C.	% of Pred. T.V.C.	One second V.C. (% of T.V.C.)	O_2 Sat. room air	O_2 Sat. with 100% O_2
P.E.	(M)	29	L. pneumonectomy	36	47	76	94	100
*C.M.	(F)	58	L. pneumonectomy	22	38	41	93	98
D.A.	(F)	27	R.U. lobe resection	29	38	70	90	100
*H.H.	(M)	35	Multiple segment resection	22	44	52	89	100
P.O.	(F)	27	L. pneumonectomy	36	47	60	85	100
L.M.	(F)	26	L. pneumonectomy	41	53	70	89	100
*W.	(M)	46	R.U. lobe and segment resection	16	45	47	91	99
N.O.	(M)	50	R.U. lobe resection	46	93	68	93	100
G.A.	(M)	34	L.U. lobe resection	57	109	60	87	100
C.M.	(F)	38	R.U. lobe resection	37	74	62	93	100
I.	(F)	38	R.U. and M. lobe resection	37	78	59	91	99
G.M.	(M)	57	L. pneumonectomy	24	68	44	91	100
B.M.	(M)	54	R. pneumonectomy	32	45	69	94	100
H.I.	(M)	60	L. pneumonectomy	61	64	86	94	100
M.O.	(F)	41	R.U. lobe resection	65	55	81	94	100
T.A.	(F)	28	L. pneumonectomy	47	50	82	93	100

M.B.C.—Maximum breathing capacity.
T.V.C.—Total vital capacity.
*Postoperative deaths.

Salvage resections. — A brief mention should be made of the so-called "salvage resections". These patients, 16 in our series, were borderline respiratory risks (Table IV). They had spent five to 15 years in the sanatorium and all were sputum positive and drug resistant at the time of operation. Three of our five deaths were in this group of patients. This gives a mortality rate of 19% compared to a mortality rate of 1.5% in the remaining cases. These patients are desperate risks and postoperative management is difficult. Here elective tracheotomy at the end of operation may be lifesaving by decreasing the dead space and by permitting pulmonary secretions to be suctioned adequately. Digitalization of the elderly patient and the use of bronchodilators to keep the lung "dry" are also necessary measures in management. Parenteral fluids must be kept to a minimum, or pulmonary œdema will be produced.

Discussion

From this group of consecutive resections for pulmonary tuberculosis performed by a senior surgeon and resident staff it is apparent that complications of pulmonary surgery in tuberculosis are not rare. Fortunately, serious complications are uncommon but when they do occur the mortality rate is high. Thus it behooves both physicians and surgeons to see that patients who could be better managed by thoracoplasty are not presented for resection.

In contrast to other reports, we have noted no relation between age or sex and the incidence of complications. The North American Indian, although more prone to contract tuberculosis, does not need more radical surgery than does his white brother. Also, Indians are no more prone to complications than whites in spite of a lesser appreciation of surgery and almost no regard for postoperative orders after the first week. In fact, if anything, they do better after operation than the white patients.

The important factors in the development of complications after pulmonary resection are the type of operation performed and the nature of the disease. Segmental resection, which leaves a raw surface of lung, is most commonly complicated, and air leak and residual pleural space are sequels. These can be minimized but not excluded by careful technique and by tailoring thoracoplasty. Tube drainage and suction have been found quite adequate in our hands, and we do not hesitate to leave an apical air pocket to absorb spontaneously. No harm has come from such a conservative approach and this has been the experience of others. With the patient on anti-tuberculosis drugs, the danger of empyema or reactivation is negligible.

When the patient's sputum is positive and resistant to the anti-tuberculosis drugs, complications, although only slightly more common, are exceedingly serious. Bronchopleural fistula in such a patient carries a 50% mortality rate. More care must be taken in these cases, but we have not found a positive sputum and drug resistance to influence our results so long as active endobronchitis is absent. This contrasts with the views of others who feel that these factors cause a higher complication rate.

Resection in pulmonary tuberculosis is a valuable procedure but must not be performed to the exclusion of all other techniques. If it is, the complication rate will bring resection into disrepute. Fortunately, serious complications from resection are rare, and will remain so when a proper balance is maintained between it and thoracoplasty. At our sanatorium the proportion is four resections to one thoracoplasty. It must be recognized that the price of a complicated resection may be too high if a simpler and safer procedure would have given the same end result.

Summary

The complications of resection in pulmonary tuberculosis in 150 consecutive operations are discussed and the management of the major complications is outlined. The North American Indian is no more prone to complications than is the white. A proper balance between resection and thoracoplasty will keep the complication rate at a reasonably low level.

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Résumé

Pendant près d'un siècle, l'unique arme dont disposait la thérapeutique chirurgicale antituberculeuse était la collapsothérapie. Avec l'apparition de la chimiothérapie, il devint possible de faire l'ablation de parties atteintes, et on peut dire qu'une exagération certaine se fit jour à ce propos. Le présent article traite des résultats et complications observés dans 148 cas (68 blancs et 80 indiens) de résection pulmonaire. Sur ce nombre, 139 malades étaient bacillaires et 46 résistants aux antibiotiques. L'intervention avait pour indication de réséquer une zone pulmonaire malade ou détruite.

L'ensemble peut être résumé comme suit: cinq cas de décès: embolie pulmonaire—œdème aigu compliquant une réaction transfusionnelle—insuffisance respiratoire—hémorragie secondaire; deux cas de fistules bronchiques dont l'une, d'origine staphylococcique, guérit après drainage et résection costale et l'autre, qui nécessita un Schede, mourut d'hémorragie secondaire (cas rapporté plus haut); deux cas d'empyèmes, un staphylococcique et un tuberculeux après pleuro-pneumonectomie gauche. Tous deux réagirent bien à la thoracoplastie. L'empyème peut naître spontanément ou résulter d'une fistule, il peut aussi provoquer celle-ci. Dans ces cas il faut essayer de fermer la fistule, drainer et s'aider de la thoracoplastie. En ce qui concerne les fistules il faut se souvenir qu'il est plus facile de prévenir que de guérir cette complication: les sutures devront toujours être faites avec grand soin sur un moignon court, bien vascularisé et bien recouvert.

Dans plus de la moitié des cas de résection il se fait des fuites d'air et une persistance de l'espace pleural, soit immédiatement après l'intervention, soit entre le cinquième et le quinzième jour. Le traitement est d'établir une aspiration continue sur le drain; ceci finit toujours par s'arranger et la thoracoplastie n'est jamais nécessaire.

Parmi les autres complications on compta: trois cas d'atélectasie, dont deux durent être bronchoscopés et l'autre subit une trachéotomie. L'atélectasie est une complication qui doit pratiquement disparaître si la physiothérapie post-opératoire est bien faite. De plus, l'aspiration des voies aériennes devra toujours être pratiquée très soigneusement à la fin de l'intervention.

Trois cas d'hémorragies dont deux réagirent favorablement à la transfusion et l'autre nécessita une nouvelle opération pour refaire une ligature artérielle qui avait dérapé. Dans les complications diverses citons un cas d'iléus paralytique, deux cas d'infection de la plaie et deux cas de fibrothorax.

Dans l'ensemble, le pourcentage de complications ne paraît lié ni au sexe, ni à l'âge, ni à la race des malades. Ce qui compte le plus, c'est le type d'intervention et la nature de la maladie; par exemple, il semble avéré que les fuites d'air sont plus fréquentes après les segmentectomies. D'autre part, il faut remarquer que lorsque les expectorations sont bacillaires et le malade résistant aux antibiotiques, les complications ne sont pas plus fréquentes, mais elles deviennent beaucoup plus graves: la fistule bronchique dans ces cas entraîne 50% de mortalité.

Si la résection est une bonne thérapeutique, elle ne doit pas être exclusive, faute de quoi le pourcentage des complications risquera d'amener son discrédit.

SOLITARY DIVERTICULITIS OF THE CÆCUM AND ASCENDING COLON*

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DIVERTICULITIS OF THE CÆCUM and ascending colon is a rather uncommon condition, clinically indistinguishable from acute appendicitis. Familiarity with the entity is essential for its recognition at operation since the lesion may resemble an inflamed carcinoma of the cæcum. The latter condition would demand extensive bowel resection, and the desirability of avoiding such a procedure in a benign inflammatory process is self evident. Fourteen proven cases of solitary diverticulitis of the cæcum have been encountered at the Montreal General Hospital since 1946. These are being presented to illustrate the clinical syndrome, the difficulties in diagnosis, and the problems in treatment.

Multiple diverticulosis of the colon is a subject familiar to all surgeons, but should not be considered related to the present subject for a number of reasons. The diverticulum we are describing is characteristically solitary and, although anatomically involving the cæcum or ascending colon, is generally referred to as "solitary diverticulum of the cæcum". Although Schnug[23] created a decided controversy when he classified all diverticula of the cæcum as congenital or acquired, it is currently considered (but certainly not proven) that these diverticula are congenital in contradistinction to the acquired multiple diverticula of the colon. Further, the clinical manifestations of this disease are different, in that bowel irregularities are not found, obstructive symptoms have never been reported, and the affected individuals are on the average more than 10 years younger than those afflicted with diverticulitis of the sigmoid.

The first case of solitary diverticulitis of the cæcum was reported by Potier[21] in 1912, occurring in a 32 year old woman operated upon for appendicitis. Subsequently, case reports have appeared with increasing frequency and these have been collected and analyzed periodically. In 1937, Bennett-Jones[3] reviewed 17 cases from the available literature and added three of his own; Baker and Carlile[2] collected 39 cases and in 1947 Anderson[1] reported on 99 cases. By 1952, the total number of reported cases stood at 126[4] and in 1952 Lauridsen and Ross[14] presented a comprehensive review of 153 cases. This has been superseded by the report of Nissenbaum *et al.*,[17] which reviewed 166 cases summarized from the literature. The present authors believe that this condition is more commonly encountered by surgeons than the above reports would indicate; this should serve to stimulate interest in a condition which should be remembered when an inflamed cæcal mass is encountered at laparotomy.

CASE REPORTS

The following case reports were summarized from the medical records of the Hospital.

CASE 1.—Mrs. O.S., 55, June 22, 1946. The patient had a history of nausea and vomiting and complained of right sided abdominal pain of 72 hours' duration. Temperature 100.3° F., pulse 100, white blood cell count 8750. There was marked tenderness in the right lower quadrant of the abdomen and a diagnosis of acute appendicitis was made. At operation, a large mass in the ascending colon with surrounding inflammatory reaction was encountered. The appendix appeared normal. The operating surgeon could not exclude carcinoma of the cæcum and a right hemicolectomy was performed. The pathological diagnosis was that of acute suppurative diverticulitis of the cæcum, and the diverticulum was located 3 cm. distal to the ileocæcal valve. The patient's postoperative course was complicated by a fistula from which she recovered and she has been well subsequently.

CASE 2.—Mr. W.McL., 49, December 21, 1958, was admitted complaining of crampy periumbilical and right lower quadrant abdominal pain of two days' duration. Temperature 100.2° F., pulse 88. There was tenderness and muscle splinting on palpation in the right lower quadrant. With a diagnosis of acute

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appendicitis, operation was performed. There was free fluid in the abdominal cavity, and the cæcum was inflamed but the appendix was not involved in this reaction. A mass was palpable in the cæcum. Carcinoma of the cæcum was diagnosed and a right hemicolectomy carried out. Pathological report: "Ulcerative and suppurative diverticulitis of the cæcum with extensive acute diffuse exudative reaction and partial infarction". The diverticulum was located on the postero-lateral wall of the cæcum. The patient recovered satisfactorily and was well when last seen.

CASE 3.—Mr. A.H., 54, May 13, 1949. Lower abdominal pain of one week's duration was the patient's presenting complaint. Temperature 97.8° F., pulse 88, white blood cell count 7500. There was some right lower quadrant tenderness on abdominal examination. The patient was thought to have chronic appendicitis and was operated upon. A mass was encountered on the medial aspect of the ascending colon 12.5 cm. from the ileo-cæcal junction. The appendix was normal. The operating surgeon considered the lesion to be either carcinoma or diverticulitis, and a right hemicolectomy was performed. The pathological diagnosis was of diverticulitis of the colon with chronic non-specific inflammatory reaction. The patient recovered satisfactorily. He has since undergone cholecystectomy for cholecystitis and subtotal gastrectomy for duodenal ulcer and currently suffers from dumping symptoms.

CASE 4.—Mr. G.W., 34, March 9, 1950, suffered from acute abdominal pain for five days before admission. Temperature 99.8°F., pulse 80. There was marked tenderness, muscle-guarding and rebound tenderness on examination of the right lower quadrant of the abdomen. Acute appendicitis was diagnosed. At operation, free purulent fluid was found in the abdominal cavity. There was an inflammatory mass 6 cm. in diameter located on the anterior aspect of the cæcum near the ileo-cæcal junction. The appendix was not inflamed. The mass was biopsied, and frozen section diagnosis confirmed the suspicion of acute cæcal diverticulitis. An attempt was made to resect the mass locally, but owing to its position near the ileo-cæcal valve this was considered impossible without jeopardizing the blood supply of the ascending colon. A right hemicolectomy with ileo-transverse colonic anastomosis was performed. The pathological diagnosis was acute diverticulitis of the cæcum. The patient's postoperative course was smooth, and he has had no further abdominal symptoms.

CASE 5.—Mrs. C.S., 36, April 17, 1951. The patient complained of intermittent crampy right lower quadrant abdominal pain associated with constipation for one year. Temperature 98.6°F., pulse 80. There was muscle guarding and some tenderness on deep palpation in the right lower quadrant. A diagnosis of chronic appendicitis was made but at operation a non-inflamed appendix with distal adhesions was encountered. Above the ileo-cæcal valve there was a diverticulum which contained fæcal material. A routine appendectomy was performed. The diverticulum was transected at its base and removed. The base was inverted into the wall of the cæcum. The pathology report described a diverticulum 2 cm. long and 1 cm. in diameter, with diverticulitis of the cæcum and a normal appendix. The patient's postoperative course was completely uneventful and she has remained well to date.

CASE 6.—Miss P.R., 25, December 7, 1952. The patient had a history of a recent chest cold and complained of loss of appetite and mild diarrhœa for 10 days, with onset of right lower abdominal pain 24 hours before admission. Temperature was 99.0° F., pulse 88, and white blood cell count 13,000. There was mild generalized resistance and acute tenderness in the right lower quadrant on abdominal examination. On deep palpation a mass was felt in the right lower quadrant. The diagnosis of acute appendicitis was made. At operation, there was a sharply demarcated mass on the posterior wall of the cæcum 4 cm. distal to the ileo-cæcal valve. There were palpable lymph nodes in the mesentery. The appendix was normal. The operator diagnosed a tumour of the cæcum and performed a right hemicolectomy. The pathologist described a 1.5 cm. diverticulum with muscle layers present in part of the wall around the ostium. There was a marked inflammatory reaction present, and diverticulitis of the ascending colon was diagnosed. The patient made an uneventful recovery and was perfectly well when last heard from in December 1957.

CASE 7.—Mr. T.R., 57, December 23, 1952. This man had complained of intermittent right lower abdominal pain for one year which became severe four days before admission. Temperature 98.0° F., pulse 88. There was

right sided tenderness, maximal in the lower quadrant, with tenderness on deep palpation. Rectal examination revealed tenderness anteriorly and on the right side. The clinical impression was one of acute appendicitis. At operation, a perforated diverticulum of the cæcum was encountered two inches (5 cm.) above the origin of the appendix with associated inflammation of the appendix. The diverticulum was transected, its base was inverted and an appendectomy was performed. The pathological diagnosis was "diverticulitis of the cæcum with chronic actively fibrosing inflammatory reaction and subacute appendicitis through involvement 'by contiguity' ". This diverticulum was situated on the medial wall of the cæcum 5 cm. above the appendix. The patient recovered uneventfully and was well at last report.

Case 8.—Mrs. T.B., 40, October 9, 1953. The patient had a history of nausea and intermittent right lower quadrant pain for three weeks. Temperature 99.2° F., pulse 72, white cell count 8800. On examination there was right sided tenderness, maximal in the lower quadrant. A diagnosis of acute appendicitis was made. Operation revealed a gangrenous diverticulum 1 cm. in diameter on the lateral wall of the cæcum below the ileo-cæcal valve. An ovarian cyst and normal appendix were also noted. The diverticulum was inverted into the wall of the cæcum, where it was retained by a purse-string suture, and a routine appendectomy was performed. The patient made a normal recovery and has since remained well.

Case 9.—Mr. D.H., 26, May 4, 1953. The patient complained of severe mid-abdominal pain which migrated to the right lower quadrant of the abdomen and was of eight hours' duration. Temperature 98.0° F., pulse 76. Marked tenderness was evident in the right lower quadrant, with slight tenderness to the right side on rectal examination. A diagnosis of acute appendicitis was made and the patient taken to the operating room. The surgeon encountered an acutely inflamed diverticulum on the medial aspect of the base of the cæcum. A retrocæcal appendix with serosal inflammation was also found. The diverticulum was transected at its base and the stump inverted by means of a purse-string suture. Appendectomy was also performed in the usual manner. The pathologist reported chronic diverticulitis of the cæcum with subacute serosal appendicitis. The patient made an uneventful recovery and has since remained well.

Case 10.—Mr. J.S., 39, September 11, 1956. There was a history of generalized abdominal pain of one week's duration with increasing severity in the right lower abdomen. Temperature 97.4° F., pulse 70 and white blood cell count 13,750. Tenderness was noted in the right lower quadrant of the abdomen, and there was a small slightly tender mass palpable in this area. Rectal examination revealed tenderness on the right. A diagnosis of acute appendicitis was made and at operation a 3 x 4 cm. mass was discovered near the base of the appendix on the postero-lateral aspect of the cæcum. Carcinoma of the cæcum was suspected but a biopsy of the mass was not diagnostic. The abdomen was closed to allow preparation of the patient for resection. Subsequently, an air-contrast barium enema examination was performed, which did not outline the cæcum satisfactorily, and the diagnosis remained carcinoma of the cæcum. On re-exploration one week later, the cæcal mass was found to be greatly reduced in size. A cæcotomy was performed and the mucosa found normal except for an area occupied by a diverticulum. The cæcotomy was closed. The appendix and diverticulum were excised, together with a wedge of cæcal wall. The defect was then repaired in two layers. The pathological diagnosis was "subsiding cæcal diverticulitis and chronic peritonitis of the appendix". The patient's recovery was uneventful and he has remained well.

Case 11.—Mrs. G.C., 41, December 22, 1956. This patient had a history of dull periumbilical pain which migrated to the right lower quadrant after three or four days. Temperature 100.6° F., pulse 122. There was marked right lower quadrant rigidity and rebound tenderness referable to this area. A diagnosis of acute appendicitis was made. At operation, an 8 x 3 cm. mass was found on the postero-medial wall of the cæcum with a deep ulcer in its centre. The mesocæcal lymph nodes were enlarged. The surgeon diagnosed carcinoma of the cæcum, possibly diverticulitis of the cæcum, and performed a right hemicolectomy. Pathological examination revealed an ulcerative diverticulitis on the medial side of the ascending colon above the ileo-cæcal valve. The ulcer was 2 cm. in diameter. The appendix was normal. The patient recovered satisfactorily and subsequently has been well.

Case 12.—Mrs. L.C., 22, February 7, 1957. The presenting complaints were of sharp colicky pain in the right lower abdomen for

one week before admission. Temperature 98.6° F., pulse 128. On examination there was minimal tenderness; bowel sounds were reduced, and a mass was palpable in the right lower quadrant. It was the clinical impression that the patient had a perforated appendix. At operation, the appendix was normal, but there was an acutely inflamed diverticulum of the cæcum occupying the anterior wall below the ileo-cæcal valve. The diverticulum was transected at its base, inverted and oversewn. Appendectomy was also performed. The pathologist's report confirmed the diagnosis of ulcerative diverticulitis of the cæcum and a normal appendix. The patient made a normal recovery and has remained well.

CASE 13.—Miss M.D., 40, March 20, 1957. The patient had steady right lower quadrant pain for four days and was admitted with a temperature of 98.4° F., pulse 90 and white blood cell count of 12,200. Examination of the abdomen revealed a 5 x 5 cm. ill-defined mass in the right lower quadrant which was also palpable rectally. It was thought that she had acute appendicitis with omental localization. At operation, a firm slightly inflamed mass was found in the lateral portion of the caput cæcum. A normal appendix and irregular uterine mass was also encountered. A diagnosis of carcinoma of the cæcum was postulated and the abdomen was closed so that the patient could be prepared for resection. A barium enema examination during convalescence was not contributory. On re-examination one week later, the mass had resolved to a marked degree and the diagnosis of cæcal diverticulitis was obvious. Colotomy was performed, the diverticulum dissected out and resected locally, and the defect closed in two layers. Appendectomy and uterine myomectomy were also performed. The pathologist reported diverticulitis of the cæcum in a 2.5 cm. x 2.5 cm. diverticulum, an appendix with focal endometriosis and leiomyomata uteri. The postoperative course was uneventful and the patient has remained well.

CASE 14.—Mrs. R.R., 37, October 23, 1957. The patient complained of right lower quadrant pain of two days' duration before coming to hospital. Her temperature was 99.2° F. and pulse 90. There was muscle splinting and tenderness to palpation on abdominal examination. Rectal examination was not remarkable. Acute appendicitis was diagnosed and an operation performed. A normal appendix was encountered, but on the antero-medial wall of the ascending colon 5 cm. above the ileo-cæcal junction, an inflamed mass was found. The ascending colon was opened opposite this mass and a diagnosis of acute diverticulitis was made when the ostium was viewed. Since the diverticulum was shallow, it was considered that oversewing the inflamed colon wall with omental fat would ensure against perforation, and healing processes would obliterate the diverticulum. This was done, the colotomy was closed and appendectomy was then performed. The appendix was found to be normal on examination. The patient's recovery was completely normal and she has remained well.

DISCUSSION

Etiology

The origin of the cæcal diverticulum remains unsettled, but many theories have been advanced to account for the solitary nature of the lesion.

Cæcal diverticula have arbitrarily been divided into the true or congenital type in which all layers of the normal wall are represented, and the false or acquired diverticulum in which one or more of the muscle layers are missing. This classification was advanced by Schnug,[23] but is not considered practical, for as Kirkman[13] has pointed out, unless serial sections are made, it is difficult to be certain whether one or more layers are actually missing. The entire wall of the diverticulum may be grossly destroyed by the associated inflammatory process and necrosis, or conversely, an eccentric section may show muscularis in a false diverticulum.[12, 13, 15]

A brief review of several of the theories advanced will serve to illustrate the inadequacy of current knowledge. Bennett-Jones[3] regarded the diverticula as acquired and supported the concept that they were the result of a weakness in the intestinal wall caused by a gap in the muscle at the entrance of blood vessels. This concept was also supported by Anderson[1] but has not withstood the critical examination of experience. Both Bennett-Jones[3] and Jonas[12] have suggested that tuberculous adenitis may be a factor in producing diverticula.

Fairbank and Rob[7] postulated that a false diverticulum could occur at the site of a solitary healed ulcer of the cæcum.

Lipton and Reisman[16] on the other hand suggest that the solitary cæcal ulcer could be the result of healing processes in an inflamed diverticulum. The solitary ulcer of the cæcum is a related subject, and there is considerable controversy over its true nature. Dorling[5] also felt that traction through healing of an ulcer could cause the false type of diverticulum, while the true types were the result of the presence of the "second appendix". Waugh[26] considered embryological aberrations as responsible for the occurrence of the "second appendix" in a case he reported, but suggested that this abnormality might really have been a solitary cæcal diverticulum. Reid[22] quotes and supports Evans[6] who suggested that irregularities in the process of vacuolation of the intestinal entoderm caused the cæcal diverticulum, in much the same manner as an intestinal reduplication develops. In support of this concept, there is the case of diverticulum of the cæcum in a three year old girl, reported by Odqvist and Petren.[18] In his recent three volume work, Shackelford[24] accepts the embryological origin of the diverticula and this concept is further supported by recent papers.[17, 19] Finally, current authors tend to favour the congenital origin of diverticula of the cæcum and support the view that the condition differs materially from and is not an extension of diverticulosis of the colon.

Pathology

Although the theories of etiology may be of interest and of ultimate importance, it is the pathological features of the condition that chiefly concern the surgeon. The diverticula are approximately 1 cm. in diameter at the ostium and vary from 1 to 3 cm. in length (0.9 x 2.7 cm.).[14] Fæcal material or fæcoliths are present in practically all cases and cause the obstruction which is believed responsible for the onset of the inflammatory reaction. Possible pathological changes have been outlined by Parker and Serjeant.[19] The acute inflammatory stage is, as expected, characterized by hyperæmia, leukocytic infiltration, suppuration, ulceration and possibly gangrene, with adjacent pericæcal œdema and peritoneal reaction. Clinically, acute appendicitis is suggested. Subsequently, the process may resolve with recurrent low grade localized infection. "Chronic appendicitis" may be suspected. On the other hand, the acute process may proceed to perforation, with either a localized abscess or generalized peritonitis resulting. If the abscess, which is commoner, is simply drained, the operator may remain under the impression that the appendix is the organ at fault. Thirdly, if the condition extends beyond the diverticulum to involve adjacent cæcum and pericæcal tissues, a large inflamed granulomatous mass with palpable mesenteric lymph nodes indistinguishable from an infected carcinoma of the cæcum confronts the surgeon at laparotomy. The appendix is frequently involved by contiguity and a periappendiceal reaction is seen. A differential diagnosis to be considered will be presented below.

In the present series, 12 specimens were submitted to the pathologist. Of these, nine were located in the cæcum and the remainder were in the ascending colon near the ileo-cæcal valve. Diverticulitis was diagnosed in all; two being acute, three ulcerative, one subsiding, and one chronic. In five cases, the stage was not stated. The presence or absence of muscularis in the sections was not uniformly commented upon, although in one report (Case 6), it was stated that all coats were present. However, we will not consider dividing the diverticula into true or false types. A photomicrograph of the diverticulum from Case 4 is presented in Fig. 1.

The location of these diverticula on the cæcum has been of passing interest to some reviewers including Schnug,[23] Lauridsen and Ross,[14] and Baker and Carlile.[2] It is stated[14] that 78% of 118 diverticula occurred within 2 cm. above or below the ileo-cæcal valve. Baker and Carlile charted 25 diverticula and found them to be of decreasing frequency on the anterior cæcal wall, the lateral wall, and the caput. The diverticulum is rarely if ever found enclosed in an appendix epiploica, in contradistinction to those found in the sigmoid colon.

The position of the diverticula recorded in the present series is charted in Fig. 2.

Diagnosis

It seems to have been practically impossible to make the diagnosis of acute solitary diverticulitis of the cæcum preoperatively – the five reported exceptions were diagnosed preoperatively by barium enema examination, two of these patients having had a previous appendectomy.[14, 17] Schnug[23] tabulated the symptoms and signs of acute diverticulitis and found them practically indistinguishable from those of acute appendicitis. Nausea and vomiting appeared to be more frequent with appendicitis than with diverticulitis. Nissenbaum, Sparks and Ellison[17] reported that a right lower quadrant mass was palpable in 50 of 148 cases. A mass was noted in four cases (Cases 6, 10, 12 and 13) in our group.

All decades from the first to the seventh are represented. The average age in all groups was 39.6 years, distinctly less than the average of 53.6 years quoted for diverticulitis of the sigmoid colon.[20] The sexes are equally affected. Age and sex incidence in the Montreal General Hospital series closely resemble those quoted by others (Table I).

The experience of surgeons in this hospital has not differed from those in other centres as regards preoperative diagnosis. Uniformly, appendicitis was suspected and operation performed. At operation, the correct diagnosis was evident in seven cases (Cases 4, 5, 7, 8, 9, 12, and 14). In the remaining seven cases a tumour was diagnosed or could not be excluded. A right hemicolectomy was performed in five of these seven cases; in the two remaining cases the abdomen was closed after exploration in order that the patient might be prepared for colonic surgery. In these two cases, at the second operation, the diagnosis of diverticulitis was evident because of the marked resolution of the inflammatory process in the interim. In a third case (Case 4), the mass was biopsied and the correct diagnosis obtained. It would seem that this procedure is attended by too many possible dangers to be recommended routinely. Thus in 7 of 14 cases, the correct diagnosis was not obtained at the first operation.

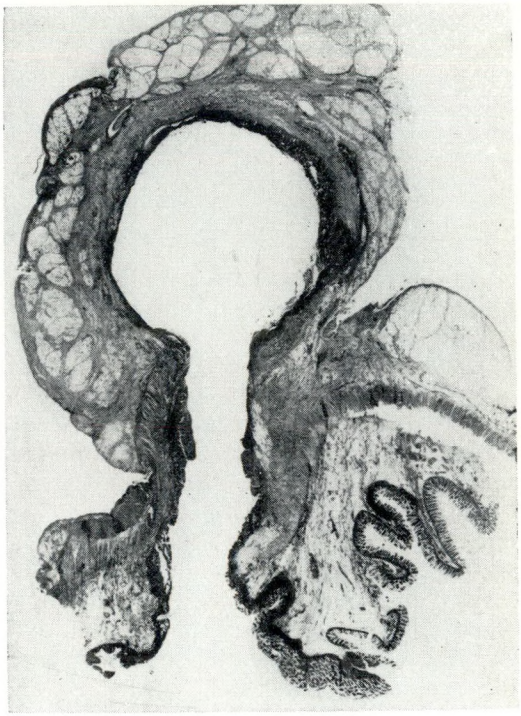

Fig. 1.

In the remaining cases, the diagnosis was evident; there was localized inflammatory reaction, ulceration, or gangrene without gross distortion of the appearance of the diverticulum. The correct diagnosis was made at the first operation in 50% of our cases as compared with 66.9% of the 133 operations reported by Lauridsen and Ross

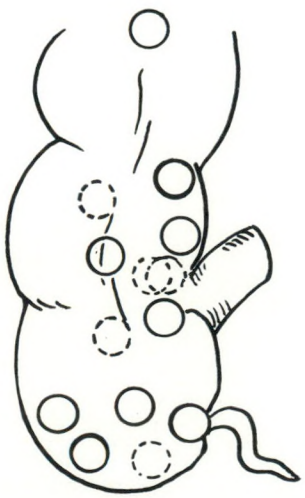

Fig. 2.

TABLE I.—Comparison of Populations

	Age		Sex		
	Range	*Average*	*Males*	*Females*	*Ratio*
M.G.H.	22 - 57 years	♂ 43.1 \ 39.6 years ♀ 37.0 /	6	8	1:1.3
Lauridsen and Ross (1952)[14]	10 - 72 years	♂ 40.4 \ 40.6 years ♀ 40.8 /	84	64	1.3:1

(Table II). With increasing experience and appreciation of the condition, it is hoped that future analyses will indicate a greater percentage of correct diagnoses at operation. It is worth while to bear in mind the operative differential diagnosis suggested by Baker and Carlile[2] – carcinoma, tuberculosis, actinomycosis, nonspecific granulomas (including regional enteritis) and the non-specific ulcer. To this might also be added the carcinoid tumour and subacute foreign body perforations of the cæcum.

To facilitate diagnosis, it has been suggested that palpation of the ostium through the opposite cæcal wall might be of value.[14, 23] In three of the cases in the present series (Cases 10, 13 and 14) the cæcum was opened and the mucosal lining inspected, this manœuvre resulting in a correct diagnosis in each instance. We feel it to be superior to the palpation method. Cæcotomy was also used to facilitate diagnosis by Unger.[25]

The presence of fæcoliths in the diverticulum sometimes produces a definite negative shadow on the abdominal radiograph. Byrne, Kallan and Bassett[4] have used this finding to recommend preoperative abdominal x-ray studies as an aid in diagnosis. They suggest that the following triad is indicative of diverticulitis of the cæcum:

1. Symptoms of right lower quadrant inflammatory disease.
2. Presence of a fæcolith in the region of the cæcum on x-ray examination.
3. History of a previous appendectomy.

Five cases have been reported in which the correct preoperative diagnosis was obtained by the use of barium enema examination.[11, 17] In the present series, two patients were examined by barium enema, but the diagnosis was not evident as the diverticula were filled with fæcal material. Since it is estimated that over one-half of diverticula contain fæcoliths,[14] one wonders whether barium enema would prove as useful a diagnostic aid as the routine anteroposterior abdominal film.

Treatment

The methods of treating diverticulitis of the cæcum have varied from the conservative regimen advocated by Leichtling[15] and Schnug[23] to the varied operative management exemplified by Table III. It is considered that continuing infection with the possibility of perforation and its attendant morbidity would contraindicate non-operative management of an inflammatory process in the right lower quadrant of the abdomen.

Operative treatments included drainage of an abscess, oversewing, inversion or excision of the diverticulum, wedge resection of the cæcum, and right hemicolectomy with primary ileo-colic anastomosis. The most direct approach was to treat the condition as one would an appendix, i.e. by clamping the base, dividing the diverticulum, then inverting the stump with a purse-string suture. This form of excision has been termed diverticulectomy and is

TABLE II.—Comparison of Preoperative and Postoperative Diagnoses

		Appendicitis	*Diverticulitis*	*Malignancy*	*Other*
M.G.H.	Preoperative (14 cases)	100%	——	——	—
	Postoperative (16 operations)	——	56.2%	43.8%	
Lauridsen and Ross[14]	Preoperative (153 cases)	85%	3.0%	3.7%	8.1%
	Postoperative (133 operations)	——	66.9%	29.3%	3.8%

TABLE III.—Comparison of Operative Procedures Used

Operation	M.G.H. series (16 operations)	Lauridsen and Ross[14] 1952 (153 operations)
1. Excision (diverticulectomy)	4	67
2. Inversion	1	10
3. Colon resection		
a. R. hemicolectomy	6	34
b. Ileo-cæcal resection	0	7
c. Cæcectomy	2	16
4. Drainage of abscess	0	8
5. Exteriorization	0	5
6. Other	1 (Oversewn mesentery)	6
7. Closure of abdomen for further investigation	2	
	16	153
Associated appendectomy	8	

to be distinguished from local resection of the diverticulum, performed by removing a wedge of uninvolved cæcal tissue at the base of the diverticulum, then closing the defect in layers. Local excision was performed in two of the present cases (Cases 10 and 13), which were originally considered to be carcinoma and in which hemicolectomy was contemplated at re-exploration. This serves to illustrate the importance of correct operative diagnosis if the morbidity and mortality associated with a needlessly radical operation are to be avoided.

The analysis presented by Lauridsen and Ross[14] reveals that hemicolectomy was performed in 34 of 153 operations for cæcal diverticulitis with a 9.4% mortality. This operation was performed in six of 16 operations in this present series, without a death. It is apparent that these are cases in which a major colonic resection could have been avoided had the true nature of the condition been realized. There are circumstances, however, when a major colonic resection is indicated because of the position of the diverticulum. It may lie at the ileocæcal junction, thus necessitating a local colonic resection; or it may project into the leaves of the mesentery in such a manner as to jeopardize the blood supply to the colon, thus necessitating a major colonic resection.[9, 12, 13] In Case 4 of the present series a major resection of cæcum and ascending colon was undertaken for technical reasons, although the correct diagnosis was appreciated. Other procedures used in the past have included simple drainage,[3] exteriorization of the cæcum,[8] and a Paul-Mikulicz resection.[13] Experience has shown them to be either inadequate or too extensive.

It is generally agreed[5, 9, 10, 13-15, 17, 24] that local excision of the lesion is the best form of treatment. The mortality in a comprehensive series[14] was 1.6% when this procedure was used. There was no mortality associated with local resection in the present series. Kirkman[13] suggests that if the lesion is gangrenous, inversion and oversewing of the lesion is acceptable. This was performed in Case 8 of the present series. Reid[22] suggests that simple inversion of a small non-inflamed diverticulum is safe.

In all cases, except those of right hemicolectomy, the appendix was removed at the time of primary treatment of the diverticulum. This did not appear to increase postoperative morbidity, and it is recommended that appendectomy accompany diverticulectomy or local excision. Gatewood[9] supports this suggestion.

Summary

Reports of acute diverticulitis of the cæcum are not common in the literature, there being fewer than 200 cases reported to date. Fourteen cases, encountered in the medical records of the Montreal General Hospital since 1946, are presented. The etiology, pathology, and diagnosis of the condition are discussed with particular emphasis on correct operative diagnosis and the most suitable treatment.

Conclusions

Acute diverticulitis of the cæcum and ascending colon occurs more commonly than standard surgical texts or the surgical literature would suggest.

It is generally not possible to distinguish the condition from acute appendicitis on a clinical basis. Suggestions are made for facilitating clinical diagnosis.

At operation, the diagnosis is evident in approximately two-thirds of cases. In the

remaining instances, it may be concealed by the presence of an inflamed mass in the cæcum usually interpreted as carcinoma.

Colotomy (or cæcotomy) will often provide a precise diagnosis.

Biopsy of the mass may confirm the diagnosis, but may give misleading information in the presence of an inflamed carcinoma and is not recommended.

Local excision of the lesion, or in some cases simple inversion of the diverticulum, constitutes the treatment of choice. Appendectomy may and should be carried out concurrently, if at all feasible.

Major colonic resections can usually be avoided if the possibility of diverticulitis of the cæcum is considered.

ACKNOWLEDGMENT

We would like to thank members of the attending staff of the Montreal General Hospital for the use of their private cases in this review, and their co-operation in furnishing us with pertinent data.

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RÉSUMÉ

La diverticulite du cæcum et du côlon droit est une affection plutôt rare qu'on ne peut cliniquement distinguer de l'appendicite aiguë. A l'opération la lésion ressemble à un cancer enflammé du cæcum. Cette affection diffère de la diverticulose commune du côlon localisée surtout à la partie descendante et au sigmoïde. Depuis le premier cas décrit par Potier en 1912, plusieurs théories ont été avancées sur la pathogénèse de cette lésion. L'opinion courante est qu'elle est probablement d'origine congénitale.

Les diverticules mesurent 1 à 3 cm. de long. La bouche d'environ 1 cm. de diamètre est souvent obstruée par des amas stercoraux ou des concrétions. Le tableau anatomopathologique est celui d'une irritation avec réaction inflammatoire comportant de l'hyperémie, de l'infiltration leucocytaire, de la suppuration, de l'ulcération, de l'œdème péricæcal, quelquefois de la gangrène et une réaction péritonéale. La perforation peut se produire amenant un abcès localisé ou une péritonite généralisée. Si l'appendice est impliqué dans le processus, on observe une réaction périappendiculaire.

La majorité des diverticules se situent à l'intérieur d'une zone s'étendant à 2 cm. en haut et en bas de la valvule iléo-cæcale (78% d'une série de 118). D'après Carlile et Baker la distribution des lésions comporte, par ordre décroissant de fréquence, la face antérieure, la face latérale et l'ampoule. On les trouve rarement dans les appendices épiploïques.

Il est quasi impossible de poser le diagnostic avant l'opération. On prétend que les nausées et les vomissements sont moins fréquents dans la diverticulite que dans l'appendicite et que dans

environ un tiers des cas on peut palper une masse dans la fosse iliaque droite. L'âge moyen de ces malades est de 39.6 ans à comparer à 53.6 ans pour ceux atteints de diverticulose du sigmoïde. Dans la série qui nous est présentée, le diagnostic ne fut évident à l'opération que dans 7 des 14 cas. Dans les sept autres, on a pensé à une tumeur, ce qui a entrainé une hémicolectomie chez cinq d'entre eux (l'abdomen fut refermé chez les deux autres en vue de la préparation à une intervention ultérieure, mais le diagnostic devint évident à la deuxième opération par la diminution de la réaction inflammatoire). Le diagnostic différentiel doit inclure la tuberculose, l'actinomycose, les granulomes comme dans l'entérite régionale, les ulcérations non spécifiques, le carcinoïde et les perforations par corps étrangers. L'inspection de la muqueuse directement par ouverture du cæcum est la manœuvre diagnostique la plus sure. La fréquence des fécalomes diminue beaucoup l'importance que l'on peut attacher au lavement baryté. Des renseignements aussi probants peuvent être obtenus de l'examen d'une plaque simple de l'abdomen ou ces fécalomes s'inscrivent comme des ombres pathologiques. La résection majeure du colon s'impose quelquefois lorsque par exemple la localisation d'un diverticule dans le mésentère met en jeu l'apport sanguin du colon. L'excision locale est cependant la meilleure forme de traitement. Elle comporte une mortalité de 1.6%. L'inversion avec enfouissement est une formule de traitement acceptable dans les lésions sphacélées. Si l'appendice n'a pas déjà été enlevé il convient de le faire au cours de l'intervention.

THYROID CANCER IN CHILDREN*

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IN RECENT YEARS there has been an increasing interest in the problem of thyroid cancer of childhood and adolescence. Until 1955, only 138 cases had been reported in the world literature. In that year Winship and Chase[1] added an additional 147 collected cases to make a total of 285, only six of these being reported from Canada. Since then, Winship[2] has stated that he now has a record of over 400 malignant thyroid tumours occurring in children.

The following series consists of a group of six children with thyroid carcinoma, all from the greater Vancouver area and all seen between 1952 and 1957 inclusive. A group of malignant thyroid tumours in adults has also been reviewed, and it has been found that children made up 5% of the total group seen in the corresponding time period.

CASE REPORTS

CASE 1.—Male, aged seven years. Symptoms: lump in left side of neck, of one month's duration. Because of "thymic enlargement", three months after birth he had been given three treatments to the thymic area on a 110 kV machine of 100 r on three days one week apart. Examination revealed an isolated nodule in the left lobe of thyroid and this was treated by local excision on April 8, 1952. The nodule was 1.5 cm. in diameter, and on pathological examination a papillary adenocarcinoma. The patient was given 3000 r in 25 days to one anterior thyroid and upper mediastinal area, beginning one week postoperatively. He was well with no evidence of recurrence five and a half years after initial treatment.

CASE 2.—Male, aged eight years. Symptoms: swelling in the left lower neck of eight months' duration; cough, one month. At the age of two to four weeks he was given a total of 500 r to the thymic area as treatment for "thymic enlargement". On examination, a large hard mass was demonstrated in the left suprasternal region extending retrosternally and posterior to the left sternomastoid. Discrete hard lymph nodes were present in the left anterior cervical region. There was x-ray evidence of tracheal compression and deviation to the right. I^{131} uptake was 15%, confined to the right lobe; there was no uptake over the thyroid mass or in the enlarged left cervical lymph nodes. Surgical treatment: left subtotal thyroidectomy and left radical neck dissection, January 14, 1957. Tumour tissue was left at the point of attachment to trachea. Pathologically: a papillary adenocarcinoma was found with extensive metastases to the left internal jugular and left supraclavicular lymph nodes with direct infiltration of fibrous tissue overlying the trachea. He was well one year postoperatively but has residual disease. He is receiving desiccated thyroid daily.

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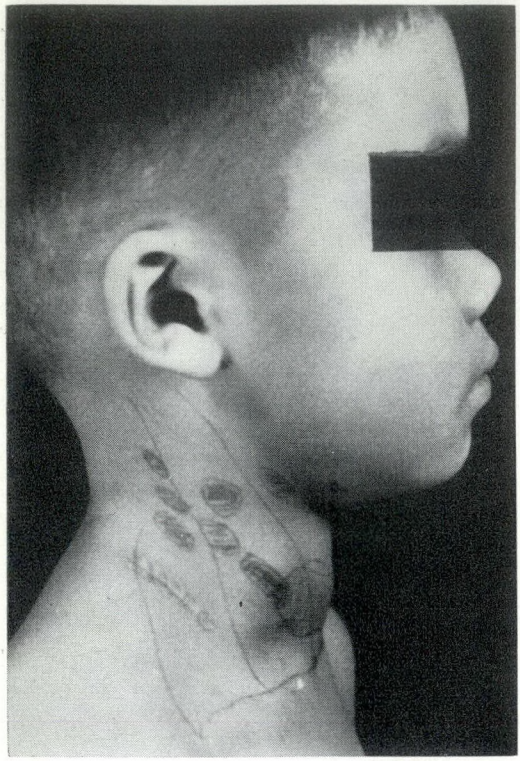

Fig. 1.—Case 3. Showing enlargement of isthmus and right thyroid lobe. Enlarged palpable lymph nodes are outlined by skin pencil markings.

CASE 3.—Chinese male child, aged eight years. He had bilaterally enlarged cervical lymph nodes and a bilaterally enlarged nodular thyroid for one and a half years (Figs. 1 and 2). He had received thymic radiation in infancy for "thymic enlargement". The dosage was 100 r a week for five doses. Extensive bilateral pulmonary metastases were also present for one and a half years before treatment (Fig. 3). A right cervical lymph node biopsy on January 30, 1956, revealed a papillary adenocarcinoma of thyroid origin. I^{131} uptake was 15% in 24 hours with 87% recovery. On scanning, no activity was noted outside the normal area of distribution of thyroid tissue. Left lobectomy and left radical neck dissection on February 29, 1956. Pathology: papillary carcinoma with extensive metastases to regional lymph nodes and small foci of blood vessel involvement. He was given a course of thyroid extract, two and a half grains per day, over a seven month period in an attempt to inhibit thyroid stimulating hormone production by the pituitary. On January 22, 1957, a right lobectomy and right radical neck dissection was performed. Pathological examination revealed a papillary adenocarcinoma with extensive metastases to lymph nodes. I^{131} uptake on February 21, 1957, three weeks postoperatively, was 6% in 24 hours and was insufficient for scanning. In an attempt to induce the chest metastases to take up I^{131}, he was given a course of three doses of 10 U.S.P. units of thyrotropic hormone intramuscularly at 12 hour intervals for three doses. Four hours after the last dose the I^{131} uptake test was repeated and showed a value of 6% in 24 hours with no appreciable uptake on scanning. A further tracer study in three weeks showed only 3% uptake. On November 4, 1957, I^{131} uptake was only 1%. He remained clinically well in spite of radiographic evidence of increased chest metastases (Fig. 4), receiving three grains of thyroid extract daily as a maintenance dose.

CASE 4.—Female, aged 12 years. Symptoms: lumps in the right side of the neck, one month's duration. Radiation treatment had been given to the thymus area for "thymic enlargement" at six to seven weeks of age. On examination she had a 1 cm. nodule in the right paracricoid area and small nodules in the right posterior triangle. Biopsy of a right cervical nodule,

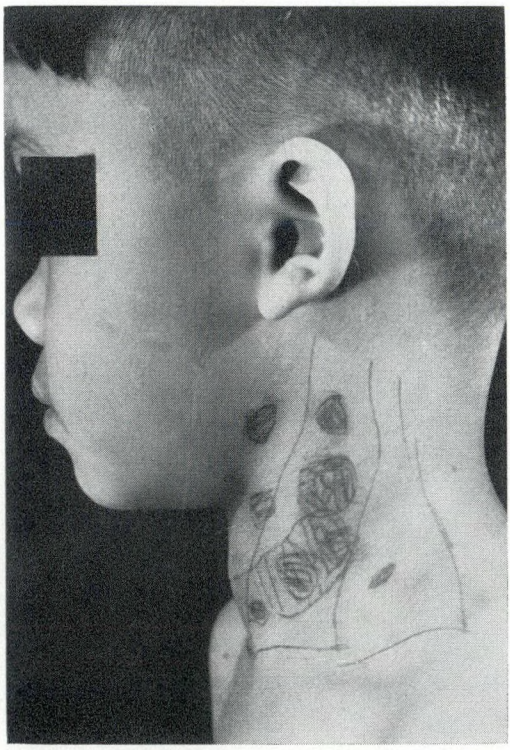

Fig. 2.—Case 3. Showing enlargement of isthmus, left thyroid lobe. Enlarged palpable lymph nodes are outlined by skin pencil markings.

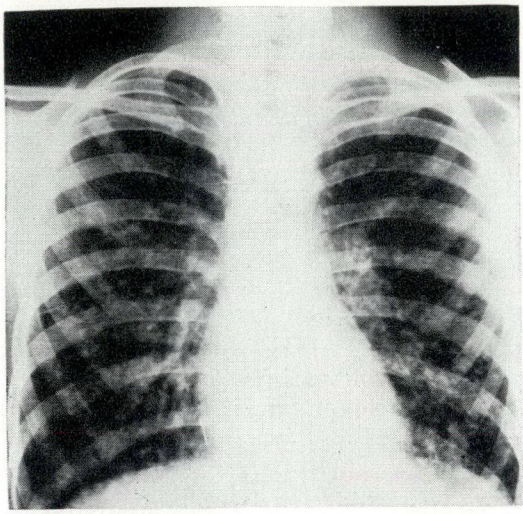

Fig. 3.—Radiographic appearance of Case 3 at the time of diagnosis, February 1956.

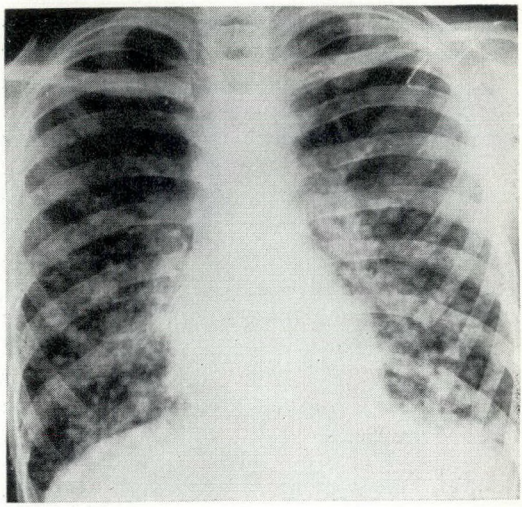

Fig. 4.—Radiographic appearance of Case 3 in November 1957.

October 18, 1956, showed a metastatic papillary adenocarcinoma. I^{131} uptake 16 days before operation was 19% in 24 hours. Total thyroidectomy and right radical neck dissection was performed on November 16, 1956. Pathologically: a papillary follicular adenocarcinoma with lymph node metastases. The uptake of I^{131} two months postoperatively was 7%. The indurated area in the right neck one year after operation may be a recurrent tumour.

Case 5.—Male, aged 14 years. Symptoms: lump in right side of neck, six months' duration. There was a history of radiation treatment for "enlarged thymus" in infancy. Examination revealed a 2 cm. x 3 cm. nodule in the right thyroid lobe, with tracheal displacement to the left. Right lobectomy was performed on January 19, 1955. Pathologically: a papillary adenocarcinoma. One year after operation, small firm nodes were demonstrated in the right side of neck. I^{131} uptake at this time showed no functioning metastases, and there was a uniform uptake in the left lobe. He was clinically well three years after operation.

Case 6.—Male, aged 17 years. Symptoms: lumps in left side of neck, six months' duration. As a young child he received 500 r over 28 days to the thymic region. Examination revealed bilaterally enlarged cervical lymph nodes and enlarged nodular left thyroid lobe. Biopsy of left posterior triangle node on July 11, 1957, showed the presence of metastatic papillary adenocarcinoma of thyroid origin. Left subtotal thyroidectomy and radical neck dissection was done on August 5, 1957. Removal was incomplete because of recurrent laryngeal nerve involvement and adherence to the trachea. I^{131} tracer uptake before operation was 21%, which on scanning was found confined to the isthmus, immediately below the thyroid cartilage. There had been no evidence of any uptake in the masses in either the right or left side of the neck. Six weeks after operation I^{131} uptake was 10%, and on scanning this was found almost entirely in the firm fixed area to the right of the midline. This suggested a definite function in metastatic tissue. On October 1, 1957, after three injections of thyroid stimulating hormone, 129 millicuries of I^{131} were given as treatment for residual thyroid carcinoma. The uptake was not accurately determined but there was considerably more taken up in the metastases than the 10%. It took five days for the level to reach 30 millicuries. There was a subsequent reduction in the size of the right sided neck mass. He was well six months postoperatively.

Comments

Clinical features.—That the condition was not readily suspected is indicated by the fact that in four instances symptoms were present for one and one-half years, eight months, six months, and five months. The two other patients had symptoms for one month each. The finding of a thyroid nodule or nodules in children is more serious than in an adult, and the majority are likely to be carcinoma.

It can be expected that the initial symptoms and signs will be the presence of

masses in the neck. As with papillary carcinomas in adults, a considerable number of patients will have metastatic lymph node involvement when they are first seen. There were four such patients in this series and one of these (Case 4) had lymph node enlargement as the only clinically demonstrable lesion. Only two of the six patients had localized thyroid nodules, both on clinical examination and at operation.

Pathology.—Histologically, five patients had a papillary adenocarcinoma while the remaining one had a papillary follicular type lesion. Papillary carcinomas are the most common type of thyroid tumour in children. That they do not always confine themselves to local spread or to localization in cervical lymph nodes is well illustrated by Case 3, in which massive pulmonary metastases were present when the diagnosis was made. Carcinoma of the thyroid is sometimes a bizarre disease in adults; that this also applies to the papillary type lesions of childhood is well illustrated by this unusual case.

Previous irradiation.—Since 1950, there have been several reports[3-6] on the incidence of previous irradiation in children who later developed cancer of the thyroid. It is extremely interesting and may be significant that all six of the patients in this series had received irradiation for an "enlarged thymus gland" during infancy. The total dosage in each instance was about 500 r. The hypothesis, advanced by Hall,[7] of an initiating factor responsible for the alteration of normal to neoplastic cells plus a promoting factor responsible for multiplication of these altered cells to form a tumour, seems applicable to this situation. This infantile irradiation might initiate occult neoplasia, the endocrine activity of childhood and puberty then promoting overt tumour formation.

Treatment.—Surgery: One patient had a local excision of a small, easily encompassed, malignant nodule in the left thyroid lobe; one a right lobectomy with complete removal of all gross disease; the remainder had excision of as much grossly involved thyroid gland and lymph node tissue as was deemed technically possible. We believe that the initial approach to differentiated thyroid cancer should be by block cancer surgery. If, after a thorough examination of the neck at operation, there is any question of lymph node spread a radical dissection is indicated. Bilateral dissections may be staged and are as applicable to childhood disease as to cancer in the adult.

X-ray: Generally, well differentiated thyroid papillary adenocarcinomas of childhood are resistant to external irradiation. Postoperative irradiation was used for two patients, one having a localized tumour, and one having more extensive lesions with residual disease. External irradiation should probably be reserved for irremovable or recurrent disease.

Radioactive iodine and hormone therapy: Five patients (Cases 2, 3, 4, 5 and 6) had tracer studies with I^{131} but only one (Case 6) showed sufficient significant uptake either in the primary lesion or in the metastases to warrant a treatment dose. These lesions were mainly papillary in type rather than follicular, and therefore a poor response was not unexpected.

In two patients, a total thyroidectomy and the administration of thyroid stimulating hormone did not induce metastases to take up I^{131} more effectively. Thyroid stimulating hormone elevation after total thyroidectomy may increase the uptake of I^{131} by metastases, and this may have been the reason for the increased avidity of the neck metastases for I^{131} postoperatively in Case 6.

There has been recent clinical evidence[8-12] that the growth of papillary thyroid cancer may be influenced by desiccated thyroid. In this small series there has been no demonstrable effect in the two patients who received courses of desiccated thyroid.

Results

The two patients with the localized disease (Cases 1 and 3) are well with no clinical evidence of recurrence, one after five years. Residual or recurrent disease is present in the other four patients.

Summary

Six cases of thyroid carcinoma in children have been presented.

The presenting symptom was usually a lump or lumps in the neck, present for several months.

All patients gave a history of thymus irradiation in infancy.

All lesions were papillary, one showing a significant follicular element. The majority had metastasized.

Treatment was primarily by surgical excision. External irradiation was used in two cases, one with locally irremovable disease. I^{131} was helpful in one case.

ACKNOWLEDGMENTS

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RÉSUMÉ

La question du cancer de la thyroïde chez les enfants a pris un regain d'actualité récemment. Jusque 1955 on ne trouve que 138 cas rapportés dans la littérature; au cours de la même année, Winship et Chase en colligèrent une série de 147.

Les auteurs présentent brièvement six cas personnels, chez lesquels les premiers symptômes étaient apparus depuis une période variant de 1 à 18 mois. Histologiquement, il s'agit le plus souvent d'adénocarcinome papillaire; les métastases lymphatiques régionales ou même pulmonaires sont fréquentes. Il est intéressant de constater que dans chacun des cas présentés, les malades avaient reçu dans leur première enfance des irradiations à dose totale de 500 r pour "hypertrophie thymique". Il y a vraiement lieu de se demander si cette radiothérapie ne serait pas reponsable de la transformation des cellules normales en cellules tumorales.

Le traitement, uniquement chirurgical, consistera en l'ablation large de la glande, et s'il y a des doutes quant aux possibilités de généralisation, l'excision des chaînes lymphatiques du cou devra être faite, au besoin des deux côtés. La radiothérapie externe ne donne que des résultats médiocres, ce type de tumeur étant peu sensible aux rayons. Il y aura lieu de la réserver pour les récidives. D'autre part l'administration d'iode radioactif est décevante, puisque ces lésions sont papillaires. Des espoirs nouveaux se sont fondés sur l'emploi de thyroïde desséchée, ce que les auteurs n'ont pu encore juger sur cette petite série de cas.

THYROID CANCER IN CHILDREN*

"Although cancer of the thyroid is not common in children, I have, during the past 20 years, treated 18 children who had thyroid carcinoma. Seventeen of the neoplasms were papillary carcinomas or follicular variants of the papillary which behave in exactly the same way as the papillary carcinomas. These tumors are not encapsulated, but involve the surrounding thyroid tissue and metastasize predominantly to regional lymph nodes. In children, papillary carcinoma may also metastasize to the lungs. The only nonpapillary carcinoma of the thyroid which I have seen in a child was in an 11-year-old girl, and it was an encapsulated angioinvasive carcinoma that grossly was indistinguishable from a benign solitary adenoma of the thyroid. The youngest child was three years old at the time of operation for carcinoma of the thyroid; the oldest was 15 years. The age distribution was uniform in the children between these ages. Eleven of the 14 children whose early histories were adequate had roentgen radiation around the head, neck, or thorax when they were infants; three had no irradiation. Six were given irradiation for thymic enlargement, two for eczema, two for enlarged adenoids or tonsils, and one for enlarged lymph nodes."

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CARCINOMA OF THE TONGUE
A STUDY OF 100 CASES AT THE MONTREAL GENERAL HOSPITAL

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Introduction

This study was undertaken to review the methods of treatment used and the results obtained in 100 patients suffering from primary carcinoma of the tongue, treated as in-patients or out-patients at the Montreal General Hospital and its Tumour Clinic. They were either private or public patients, the great majority being in the latter group. Most of the records reviewed were in the period from 1950 to 1957 inclusive; 15 were prior to 1950. The 1950 to 1957 period included all cases registered at the hospital, the follow-up being complete. The 15 prior to 1950 were selected out of a larger number because their follow-up also was complete.

Incidence of Carcinoma of the Tongue

In a five-year period, from 1953 to 1957 inclusive, a total of 3596 new cases of carcinoma were registered at the Montreal General Hospital, and of these 57 were cases of carcinoma of the tongue, i.e. an incidence of 1.58%. In the Province of Quebec, during the years 1952 to 1956 inclusive, there were 25,246 deaths from cancer of all kinds, and during the same period there were 176 deaths from cancer of the tongue, an incidence of 0.69%.† During this period, too, the Dominion Bureau of Statistics reported 529 deaths from cancer of the tongue.‡

Staging and Results

In this study, the anatomical grouping was as follows: anterior third, middle third, posterior third, dorsum and diffuse throughout the tongue. In addition to location, the lesions were staged after the method of Harnet,[1] who distinguishes Stages 1, 2, 3a, 3b, and 4:

Stage 1—Lesions confined to the tongue.

Stage 2—Lesions spread beyond the tongue.

Stage 3a—Lesions confined to the tongue but with positive neck nodes.

Stage 3b—Lesions beyond the tongue and with positive neck nodes.

Stage 4—Widespread, i.e. beyond the neck.

The type of treatment (or combination of treatments) was noted, both for the primary lesion and for the metastatic nodes when present. The pathological findings were recorded as well as the age, the sex, and the survival time in years.

In analyzing the results of treatment the following categories were used:

1. Alive and free from disease.
2. Alive with recurrence.
3. Dead from primary carcinoma and/or secondary spread.
4. Dead from other causes.
5. Unknown.

The functional results were classified as good, fair, and poor.

Treatment Policy

It is the practice in this hospital to discuss each patient at a weekly tumour conference before treatment. The departments of general surgery, plastic surgery, otolaryngology, internal medicine, radiotherapy and pathology are represented at such conferences. A full and free discussion is carried out, and a program of treatment is planned and generally adhered to.

It is recognized that a policy for management of carcinoma of the tongue can be laid down, but deviations from this policy may be dictated by special circumstances, such as the general health of the patient, disagreement among the conference group as to the stage of the disease, or failure of the patient to co-operate.

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†The authors wish to thank Mr. Paul Parrot, Department of Health, Division of Demography, Quebec Government.

‡The authors wish to thank Mr. H. G. Page, Chief, Vital Statistics Division, Dominion Bureau of Statistics, Ottawa, Canada.

The policy has been to treat with radiation those lesions confined to the tongue and those which, though involving the floor of the mouth, do not involve the alveolar mucosa. All lesions in the posterior third of the tongue are treated by radiation because of the recognized greater radiosensitivity of lesions arising in this area.[2]

A number of the smaller tumours of the anterior and middle third of the tongue are removed by wide local surgical excision. When the tumour is of such a size as to involve the floor of the mouth and extend to and involve the alveolus, primary operation is frequently used. This operation was first carried out in this hospital in 1948. This is the so-called commando operation,[3] which consists of a hemi-glossectomy, a hemi-mandibulectomy, and a radical neck dissection on the same side, with a temporary tracheotomy. This is the operation that is classified as "block surgery".

The radiation method of treatment of the primary tumour in the anterior two-thirds of the tongue may be by radium implant alone in the smaller lesions (7000 r in six days). External irradiation and radium to doses of 7000 r or 8000 r n six to eight weeks) are used in the more extensive lesions for greater fractionation and because we believe that preliminary irradiation inhibits dissemination of the disease by the implant technique.[4, 5]

In the carcinomas of the posterior third of the tongue, irradiation is used to include nodes in the cross-fire technique, since tumours in this area have a demonstrably higher radiosensitivity. Supervoltage therapy (cobalt 60) has now superseded the radium implant technique for treating these lesions in the posterior third of the tongue.

Metastatic nodes, when palpable, are often given irradiation at the time of treatment of the primary tumour, if they are in the field of irradiation. When the primary carcinoma has been controlled, the nodes are removed by a radical neck dissection, i.e. removal of the whole lymphatic area on one side of the neck, to include the sternomastoid, investing layers of fascia, carotid sheath, internal jugular vein and related lymph nodes and salivary glands of the submental and submaxillary triangles. Prophylactic lymph node excisions are not carried out except in those patients with Stage 2 lesions, who are treated by radical surgical excision.

Recurrences after irradiation are treated surgically. Recurrences after operation are treated by irradiation.

In 11 of our patients, block surgical excision of half the tongue and half the mandible was carried out, and the nodes of the neck were removed. This treatment was used either alone or after previous irradiation. Five of these patients were in Stage 1, two in Stage 2, one in Stage 3a, and three in Stage 3b. In spite of this rather small number, it is our opinion that this type of treatment is a useful surgical approach on account of the low mortality (none in this group), the reasonably good cosmetic and functional result, and the shortness of the hospital stay.

Conspectus

Location

The location of the carcinoma of the tongue was as follows: 12 in the anterior third; 42 in the middle third; 39 in the posterior third; seven on the dorsum or diffusely spread throughout the tongue.

Sex

The male predominated, 83 males to 17 females.

Age

From Fig. 1, it can be seen that the majority, i.e. 63%, were in the age group from 60 to 80 years.

Duration of Symptoms (Fig. 2)

The minimum unit of time used was one month, and the maximum was four years or more. Of the group, 83% had a history of six months or less for duration of symptoms.

Stage of the Disease

The distribution was as follows: Stage 1—54; Stage 2—six; Stage 3a—15; Stage 3b—23; Stage 4—0; Unknown—two.

Our findings confirm those of other investigators, namely, that those lesions arising from the posterior portion of the tongue are more apt to spread to the adjacent

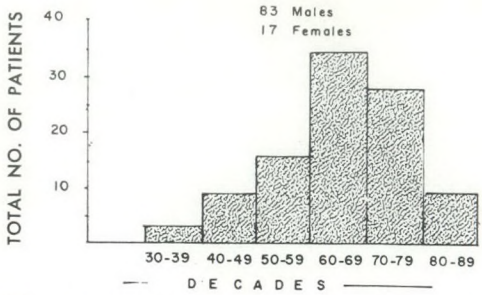

Fig. 1.—Age distribution of 100 patients with carcinoma of the tongue.

structures in the mouth or to the neck lymph nodes.[6] For example, in the anterior third, all 12 were Stage 1; in the middle third, 24 out of a total of 42 were Stage 1; and in the posterior third only 15 out of a total of 39 were Stage 1.

Predisposing Factors

A history of previous or concurrent leukoplakia was noted in 11 of the 100 patients. The use of tobacco was noted in 51. Only seven patients had a positive Wassermann reaction.

Course of the Disease

In discussing survival time, one year is the minimal measurement used. A patient marked as dying in one year lived a year or less.

Of the 12 patients with a lesion of the anterior third of the tongue, seven were alive and free of disease at the time of this study; one had died of recurrent carcinoma and four of unrelated causes (Table I).

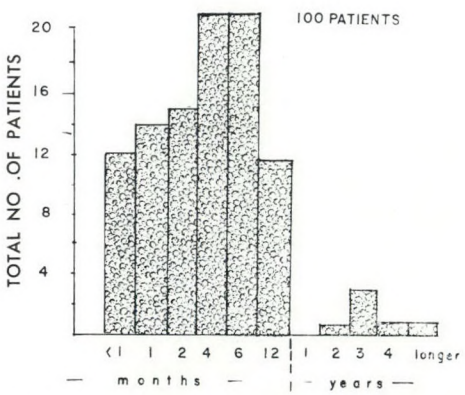

Fig. 2.—Duration of symptoms of 100 patients with carcinoma of the tongue.

TABLE I.—CANCER OF THE TONGUE: ANTERIOR THIRD—12 CASES

Alive and free from disease:
- 3—Radium; + 5 years.
- 2—Local surgery; 3 and 5 years.
- 1—Block surgery; 3 years.
- 1—X-ray and radium; −1 year.

Dead from cancer: 1
- 1—Radium, x-ray and radical surgery.

Dead from other causes: 4
- 1—Bronchopneumonia; 2 years.
- 1—Coronary disease; 1 year.
- 1—Lost after 8 years.
- 1—Presumed dead.

Of the 42 patients with middle third lesions, 15 were alive and free of disease; 19 had died of the primary disease; seven had died of unrelated causes; and in one patient the result was unknown (Table II). Of the 39 patients with posterior third lesions, nine were alive and free of disease; three were alive with recurrence; 20 had died from the disease; and the remainder had died of other causes. Of the seven patients with carcinoma of the dorsum or diffuse involvement, all were dead, but one had lived for three years,

TABLE II.—CANCER OF THE TONGUE MIDDLE THIRD—42 CASES

No. of patients	*Survival time*	*Type of treatment*
Alive and free from disease: 15		
6	5 years or longer:	1 Radium. 1 X-ray. 1 X-ray and local surgery. 1 X-ray and radium. 1 Radium and local surgery. 1 X-ray, radium and local surgery.
3	3 years:	1 Radium and local surgery. 1 X-ray, radium and block surgery. 1 X-ray and radium.
5	2 years:	3 X-ray and radium. 1 Local surgery. 1 Radium.
1	1 year:	Radium.
Dead from cancer: 19		
5	5 years or longer:	3 Radium. 1 Radium and local surgery. 1 X-ray and radium.
4	2 years or longer:	3 X-ray and radium. 1 Block surgery.
10	1 year or less:	3 X-ray. 3 X-ray and radium. 1 Block surgery. 2 Local surgery. 1 No therapy.
Dead from other causes: 8		

one for two years, two for one year and two died of other causes. The seventh patient, who had a carcinoma *in situ* of the tongue, had died of carcinoma of the œsophagus (Table IV). In the entire group of 100 patients, 60 had local control with no evidence of local recurrence of carcinoma of the tongue.

TABLE III.—CANCER OF THE TONGUE: POSTERIOR THIRD—39 CASES

No. of patients	*Survival time*	*Type of treatment*
Alive and free from disease: 9		
5	5 years or longer:	2 X-ray. 1 Radium and radical neck dissection. 1 Local surgery. 1 Radium and local surgery.
1	3 years:	X-ray, radium and block surgery.
1	2 years:	Radium.
2	1 year:	2 Block surgery.
Alive with recurrence: 3		
1	5 years:	X-ray and radium.
2	1 year:	2 X-ray.
Dead from primary carcinoma: 20		
1	6 years:	X-ray, radium, surgery.
3	2 years:	2 X-ray. 1 X-ray and local surgery.
16	1 year:	9 X-ray. 4 X-ray and radium. 1 X-ray and local surgery. 1 Block surgery. 1 No therapy.
Dead from other causes: 7		

TABLE IV.—CANCER OF THE TONGUE: DORSUM AND DIFFUSE—7

Dorsum or diffuse—6	
Dead from cancer 4	1 Radium and surgery; 3 years. 1 Radium and surgery; 2 years. 1 X-ray and radium; 1 year. 1 Local and block surgery; 1 year.
Dead from other causes 2	
Diffuse cancer of the tongue 1	1 X-ray and radium; 2½ months.

Criteria for Local Control

The ulcer or visible tumour of the tongue had disappeared following treatment and had not recurred or, if ulceration was present, microscopic examination showed it to be due to causes other than carcinoma.[7]

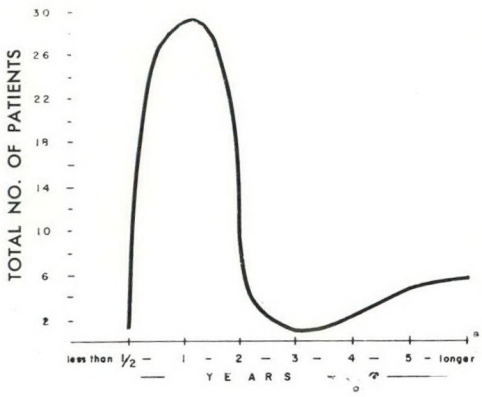

Fig. 3.—Mortality curve.

OBSERVATIONS

The plotting of known deaths of patients in this series demonstrates that the great majority of the 50 patients who have died did so within one year. The death rate levels off at the third and fourth years (Fig. 3). This figure is significant because it shows that a patient who is alive three years after treatment has a vastly greater chance of being cured of the disease. Kremen[2] stated that in his experience those patients clinically free of the disease at the end of two years were cured.

An interesting observation in this study is the high incidence of multiple carcinomas in spite of the overall short survival time. There were eight patients with second carcinomas, involving the alveolus, epiglottis, œsophagus, bladder, penis, rectum and bronchus, and one patient had a cervical lymphosarcoma in addition to his carcinoma of the tongue.

In May 1955, cobalt 60 teletherapy was first used, and in 1948 en bloc dissection techniques were first employed for the tongue. We feel that these newer techniques are advances in the treatment of the disease, but they are too recent and the number of cases is too small for numerical evaluation.

CONCLUSION

Carcinoma of the tongue made up 1.58% of all carcinomas at the Montreal General Hospital over a five-year period, 1953-1957.

When a patient with carcinoma of the tongue survives three years, his chances of

cure are good, but 35% of the patients in this series were dead within a year.

Local control is enhanced by the newer techniques of radiation therapy, 60% being the local control rate in this series.

En bloc surgical excision appears to be gaining a place in the primary treatment of selected cases of carcinoma of the tongue.

The high incidence of multiple carcinomas indicates the need for careful pretreatment evaluation and a thoughtful follow-up examination.

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Résumé

Dans cette série de cent cas de cancer primaire de la langue traités à l'Hôpital Général de Montréal, 15 seulement avaient été vus avant la période de 1950 à 1957. La fréquence de cette affection s'établit à 1.58% des cas de néoplasme découverts chaque année et à 0.69% des morts causées par le cancer. Les auteurs ont adopté la classification de Harnet basée sur la localisation aux tiers antérieur, moyen ou postérieur, au dos de la langue ou à l'organe entier. Les résultats du traitement furent évalués selon qu'il y eut survie avec ou sans récidive, ou mortalité causée soit par la tumeur primaire ou ses métastases, ou soit par une autre raison. La fonction fut estimée bonne, passable ou médiocre.

La conduite du traitement à cet hôpital est déterminée par l'opinion conjointe de plusieurs spécialistes; elle est donc apte à varier selon les cas. En général les lésions confinées à la langue, au plancher de la bouche ou au tiers postérieur sont irradiées soit par radiumpuncture des petites tumeurs donnant 7,000 r en six jours ou encore par des implants accompagnés de radiation externe donnant 7,000 à 8,000 r en six à huit semaines. La thérapie au Cobalt est en voie de remplacer l'emploi de ces implants. Les petites tumeurs du tiers antérieur ou moyen sont enlevées par résection large. Dans les autres cas l'intervention surnommée "commando" et pratiquée pour la première fois dans ce centre en 1948 est exécutée. Elle consiste en une hémiglossectomie, hémimandibulectomie et dissection radicale de la moitié ipsilatérale du cou, avec trachéotomie temporaire. La résection préventive des ganglions du cou n'est pratiquée qu'à partir des lésions du stage II. Les récidives qui suivent l'emploi exclusif de la chirurgie ou des rayons-x sont traitées par l'autre forme de thérapie à laquelle on n'a pas encore eu recours dans ce cas.

L'exérèse en bloc fut pratiquée chez 11 malades avec une mortalité nulle et des résultats dont l'apparence et la fonction sont satisfaisantes. Les lésions du tiers postérieur montrent une plus forte tendance à envahir les régions adjacentes et à s'étendre aux chaînes lymphatiques du cou, que les autres. La grande majorité des 50 morts du groupe est survenue dans l'année qui suivit le traitement. Une survie de trois ans équivaudrait donc presque à une garantie guérison. On doit noter dans ce groupe le grand nombre de néoplasmes multiples (épiglotte, œsophage, vessie, pénis, rectum, bronches) que l'on doit considérer dans le pronostic des cas de cette série.

LYMPH NODE METASTASES FROM EPITHELIOMA OF FACE, LIPS AND MOUTH

In the state of Victoria, Australia, the vast majority of patients with epithelioma of the lip, tongue and oral cavity are treated by radiation, this policy being justified by the relative infrequency of cervical node metastases, mandibular involvement or local recurrence, according to Eddey (*Austral. N. Z. J. Surg.*, 28: 34, 1958).

He managed to collect 48 patients with cervical lymph node metastases from epitheliomas of face, lip, tongue, oral mucosa, palate and tonsil; only 10 of these had undergone primary surgery, the rest having had radiotherapy. In all but two cases in the series, metastases had appeared within an average time of 3.6 months; it is probable that a patient who has survived two years without metastases after his primary treatment will remain well.

Treatment of cases with metastases by radical neck dissection in the period 1947-1955 gave a salvage rate of 37.5%. However of the eight patients who required a more extensive neck and mouth dissection in the same period, five are dead. Age is no bar to surgical treatment of such metastases.

EXPERIMENTAL SURGERY

ACID-BASE CHANGES IN DOGS WITH AND WITHOUT EXTRACORPOREAL CIRCULATION*

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THIS REPORT is concerned with the pH and pCO_2 changes in the blood that occurred as a result of extracorporeal circulation experiments in 10 dogs previously reported,[1] and in addition in 10 anæsthetized dogs without extracorporeal circulation.

Spreng *et al.* in 1952[2] perfused dogs with a heart lung machine at an average flow rate of 97 ml./kg./minute. It was observed that the pH of the blood remained relatively normal during the by-pass but dropped after perfusion; the blood pCO_2 dropped during the by-pass and rose early in the post-perfusion period.

Andreasen and Watson in 1952[3] demonstrated that a dog could survive periods up to 45 minutes when the venæ cavæ were occluded and only the blood through the azygos vein was permitted to return to the heart. This led to interest in determining the acid-base changes that occurred when the body tissues were perfused at subnormal rates.

Cohen, Warden and Lillehei in 1954[4] reported their results in 50 dogs during autogenous lobe oxygenation with perfusion rates between 8 and 14 ml./kg./minute. At the end of 30 minutes of by-pass, the pH dropped an average of 0.35 unit in nine animals, and the lactic acid level rose 31.3 mg. per 100 ml. in two animals.

It appeared at the time of this investigation that reports were lacking in the literature as regards the effects of different perfusion rates. In view of the acidotic trend as a result of perfusion, it was decided to investigate this aspect by measuring the changes in the pH and the CO_2 content in arterial blood of perfused dogs.

METHODS

Details of the perfusion techniques were as reported previously.[1] All dogs reported in this study were anæsthetized with 30 mg. sodium pentobarbital per kg. body weight.

All pH and CO_2 determinations were made on arterial blood, which was taken in oiled syringes and packed in ice. The pre-perfusion sample was taken just before the commencement of by-pass by means of a metal T connector interposed in the arterial line just proximal to the femoral catheter. The syringe was allowed to fill from the catheter after it had been flushed out with fresh blood from the perfusion animal. Sampling during perfusion was done at five minute intervals from the same site. After perfusion and during closure of the chest incision, no samples were obtained. A small catheter was left in the femoral artery after the perfusion arterial inflow catheter had been removed. The first post-perfusion sample was removed five to eight minutes after chest closure and when the dog was breathing spontaneously. At the time of each sample the temperature of either the dog or the oxygenator was noted, depending on where the blood was obtained.

The perfusion dogs were divided into a low flow rate group and a high flow rate group, perfused at the rates of 35 ml./kg./minute and 70 ml./kg./minute, respectively.

The arterial pH and pCO_2 were also followed up in two control groups. One consisted of five dogs, in which a thoracotomy of one-half hour's duration was performed to correspond to the perfusion interval in the 10 dogs which had extracorporeal by-passes. The dog's lungs during the thoracotomy period were inflated with an intermittent positive pressure respirator using compressed air. Samples were taken during this time at five minute intervals. The right chest incision was then closed and the samples were again obtained at times corresponding to the times at which samples were taken in the post-perfusion period of the perfused group of animals.

The other control group consisted of five anæsthetized dogs; no operative procedure was carried out and the dogs were allowed to breathe spontaneously. Arterial samples were

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taken every hour for five hours during the period of anæsthesia.

The pH was determined on a direct-reading Photovolt pH meter, Model 125 B, with the 2½ inch Photovolt glass electrode in an open 2 ml. cup. Commercial buffers of pH 4.0 and pH 7.4 were used to standardize the meter before each experiment. The samples were removed from the ice and the syringes allowed to warm up to room temperature for 15 to 20 minutes. The syringes remained iced for an average of one to one and a half hours. After warming, the pH determinations were then done at room temperature, utilizing the average temperature correction factor of 0.014.[5, 6] This factor was multiplied by the difference between the original temperature of the blood and the room temperature; this value was then subtracted from the reading obtained on the pH meter. This result gave the corrected or absolute value for the pH.

The remainder of the blood in the syringes was then used to determine the corresponding total CO_2 contents, utilizing the Kopp-Natelson[7] Microgasometer, Model 600, which adapts to micro-methods the classical method of Van Slyke-Neill.[8] The result was expressed in mM. per litre.

The partial pressure of carbon dioxide for each arterial sample was obtained from the Henderson-Hasselbalch equation. The value for pK′ was calculated from the nomogram reported by Severinghaus.[9] The solubility factor S for CO_2 was also obtained from Severinghaus.[10] The corrected values for pK′, S and pH, and the CO_2 in mM/l. were then substituted in the Henderson-Hasselbalch equation[10] and the result gave the pCO_2 in mm. Hg.

Standard deviations were calculated taking into consideration the small number of samples. Significance between the mean pH values of the low flow group and the high flow group at the 60 and 90 minute intervals was determined by the method outlined in Kenney and Keeping,[11] for the difference of means for small samples.

Results

Tables I and II give the mean values of pH, CO_2 content and the calculated pCO_2 content and the calculated pCO_2 at the respective intervals. The results are graphically illustrated in Fig. 1. Although the samples were taken every five minutes during the perfusion and thoracotomy periods, only the 10 minute values are reported, because the five minute plot did not alter the picture. It is observed that

TABLE I.—Effects of High and Low Blood Flow Rates During Extracorporeal Circulation in Dogs

Index *Low flow rate group (35 ml./kg./min.)*	*Pre-perfusion period*	*Perfusion period*				*Post-perfusion period*		
		0	*10*	*20*	*30*	*60*	*90*	*160*	*190*
pH	7.54 ± 0.02 (4)	7.40 ± 0.05 (5)	7.52 ± 0.06 (5)	7.50 ± 0.07 (5)	7.47 ± 0.05 (5)	7.30 ± 0.79*(4)	7.30 ± 0.09*(3)	7.38 ± 0.03 (3)	7.36 ± 0.01 (3)
CO_2 content	15.9 ± 4.0 (4)	19.8 ± 2.3 (5)	14.7 ± 2.1 (5)	13.6 ± 2.2 (5)	13.4 ± 4.2 (5)	14.4 ± 1.4 (4)	16.1 ± 0.9 (3)	19.8 ± 1.2 (3)	19.3 ± 2.2 (3)
pCO_2	18.0 ± 6.8 (4)	29.2 ± 1.5 (5)	17.0 ± 3.9 (5)	16.5 ± 3.8 (5)	17.2 ± 3.2 (5)	22.8 ± 3.9 (4)	29.0 ± 7.1 (3)	30.7 ± 3.3 (3)	27.2 ± 7.6 (3)
High flow rate group (70 ml./kg./min.)									
pH	7.45 ± 0.06 (4)	7.47 ± 0.05 (5)	7.55 ± 0.07 (5)	7.53 ± 0.10 (5)	7.51 ± 0.08 (5)	7.41 ± 0.08*(3)	7.46 ± 0.08*(4)	7.42 ± 0.05 (5)	
CO_2 content	19.8 ± 1.3 (3)	16.92 ± 3.6 (4)	15.4 ± 3.4 (4)	13.4 ± 2.2 (4)	13.4 ± 2.5 (4)	19.2 ± 0.14 (2)	16.5 ± 2.9 (3)	19.2 ± 1.7 (4)	
pCO_2	28.7 ± 4.5 (3)	21.3 ± 4.4 (4)	15.9 ± 5.5 (4)	16.1 ± 5.4 (4)	16.7 ± 5.5 (4)	30.8 ± 5.5 (2)	22.4 ± 8.0 (3)	28.0 ± 5.0 (4)	

*P > 0.05

TABLE II.—Changes in Thoracotomized and Non-Thoracotomized Dogs Without Perfusion

Index Thoracotomy control group	*Pre-thoracotomy period*	*0*	*Thoracotomy period* 10	20	30	*Post-thoracotomy period* 60	90	160
pH	7.29 ± 0.33 (7)	7.44 ± 0.04 (5)	7.50 ± 0.09 (5)	7.51 ± 0.14 (5)	7.52 ± 0.07 (5)	7.34 ± 0.1 (5)	7.36 ± 0.07 (5)	7.39 ± 0.04 (5)
CO_2 content	17.5 ± 2.3 (7)	13.3 ± 3.4 (5)	12.1 ± 1.5 (5)	12.3 ± 1.4 (5)	11.5 ± 1.4 (5)	13.7 ± 1.6 (5)	13.1 ± 3.1 (5)	13.5 ± 1.5 (5)
pCO_2	35.0 ± 6.6 (7)	19.4 ± 6.0 (5)	15.1 ± 7.1 (5)	14.7 ± 1.4 (5)	13.5 ± 1.8 (5)	24.5 ± 4.8 (5)	22.4 ± 6.8 (5)	21.5 ± 4.5 (5)
Non-thoracotomy control group	*Early post-anæsthetic period*	*60*	*120*	*180*	*240*	*300*		
pH	7.28 ± 0.05 (5)	7.31 ± 0.02 (5)	7.31 ± 0.02 (5)	7.37 ± 0.03 (5)	7.37 ± 0.04 (5)	7.35 ± 0.01 (2)		
CO_2 content	21.1 ± 2.8 (5)	20.7 ± 1.0 (5)	19.4 ± 0.8 (5)	20.6 ± 1.34 (5)	20.9 ± 0.8 (5)	19.5 ± 3.5 (2)		
pCO_2	43.8 ± 7.8 (5)	39.2 ± 3.4 (5)	36.6 ± 5.3 (5)	36.1 ± 3.0 (5)	36.2 ± 2.1 (5)	33.4 ± 5.7 (2)		

Legend for Tables I and II.

1. All values are shown as mean ± S.D.
2. ( ) Number of animals in group.
3. pH - - - - - - pH of whole arterial blood—absolute units in all instances.
4. CO_2 content - - - - - - Total carbon dioxide content in mM./l. determined from plasma of arterial blood on which pH values were determined.
5. pCO_2 - - - - - - the partial pressure of carbon dioxide in mm. Hg pressure, calculated from the Henderson-Hasselbalch equation. See Methods.

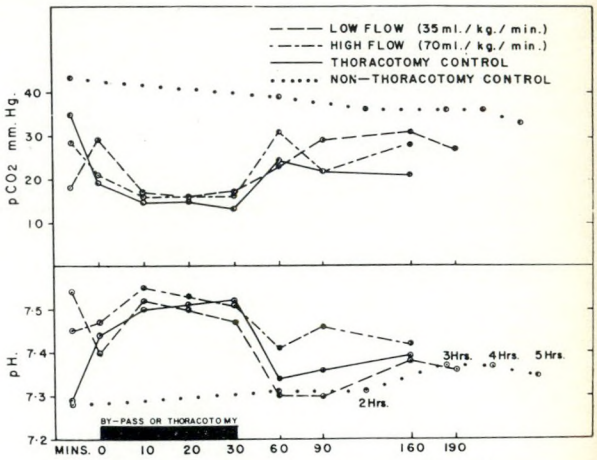

Fig. 1.—Mean values of arterial blood.

the same degree of alkalosis developed in both the low flow rate group and the high flow rate group of animals perfused as in the control thoracotomy group during the periods of perfusion or thoracotomy. There appeared to be better correlation of the pH and pCO_2 curves during this interval than at any other time during the experiment.

However, after perfusion the group perfused with low flow rates had lower values for pH, especially at the 60 and 90 minute intervals. There was no statistically significant difference in pH at these two intervals between the groups perfused at low flow and high flow rates. It was further noted that the pH values appeared to return toward normal values by 160 and 190 minutes in both groups.

In the non-thoracotomy control group the anæsthetic depressed the respiration rate, more noticeably during the first two hours, as evidenced by the average respiration rate taken hourly, as follows: 12, 16, 17, 21, 17 and 22. The anæsthetic and/or the depressed respiration may have been responsible for the low pH and high pCO_2 values in this group. The lowest pH values in the low flow rate group were observed to be no lower than the pH values in this non-thoracotomy group of dogs.

Discussion

Others have observed that an alkalosis develops during by-pass with a bubble oxygenator. Gott *et al.*[12] reported that after

a one hour by-pass with the sheet oxygenator, the average arterial pH in 15 animals was 7.48. In this study, the average pH for a 30 minute perfusion was 7.47 and 7.51 in the low flow and high flow groups respectively.

The alkalosis during perfusion, as evidenced by the high pH and low pCO_2, was no doubt due to the use of pure oxygen. However, it may be safer to have a degree of alkalosis[13] than to risk a respiratory acidosis by bubbling a mixture of oxygen and carbon dioxide through the blood during the oxygenation process.

The mean 30 min. and 60 min. post-perfusion pH value of 7.3 in the low flow group hardly reached acidotic levels, and for dogs may even be at the lower limits of normal pH range.[14]

It was realized that there were pitfalls in determining blood pH and total CO_2, especially the former. Throughout the experiment, the methods were consistently performed and it was felt that the results depicted the relative changes in these two determinations under the experimental conditions described. When using a non-aerobic glass electrode, an error could be anticipated, but the pH reading was obtained quickly and it was assumed that the escape of CO_2 during this interval was negligible. Another difficulty that arose in the measurement of blood pH was whether to attempt the determinations at 37° C. or at room temperature employing a correction factor. In the perfusion experiments, the temperature of the arterial samples varied between 33° and 39° C., so that a temperature correction factor would have been necessary even with determinations performed at 37° C.

Paneth *et al.* in their report in 1957 expressed flow rate in relation to surface area rather than to the weight of the animal. In the 10 animals perfused in this series, the weight of each animal was close to 14 kg. ± 0.9 kg., with the exception of one animal which weighed 17 kg. By calculating the surface area,* using weight equal to 14 kg., the rates for the low and high flow groups are easily converted to l./sq. metre/minute, and become 0.8 l./sq. m./minute and 1.6 l./sq. m./minute respectively. In the opinion of Paneth and his colleagues, 1.2 l./sq. m./minute was the minimum rate for adequate whole-body perfusion. According to this interpretation, the low flow group in this report falls below the minimum rate. However, the rate of 35 ml./kg./min. or 0.8 l./sq. m./min. for the low flow group was apparently sufficient, but not necessarily adequate. The evidence for this was the 100% survival of the dogs, and the absence of any severe alteration in the pH during the perfusion or in the post-perfusion period.

The alkalosis during by-pass, in the absence of determinations of fixed acid as lactic acid, makes the diagnosis of compensated metabolic acidosis impossible. It was observed that for one hour after thoracotomy in the thoracotomy control group the pH dropped to almost the same level as in the low flow group during the same period, and in both groups the pCO_2 was between 20 and 30 mm. Hg. This suggests that some other factor was responsible for the pH drop, possibly fixed acids in both the low flow and thoracotomy control groups. It seemed that for the pH to drop to the 7.3 level solely as a result of the pCO_2 effect, as for instance in the non-thoracotomy control group, the pCO_2 had to be between 38 and 43 mm. Hg in these anæsthetized dogs; this is, of course, assuming that there was no fixed acid formation in this group.

It would appear that the course after perfusion cannot be predicted from changes in pH and pCO_2 during by-pass or at the end of by-pass. If different flow rates are employed, their differential effects may not become evident until the early post-perfusion period. It is possible that the first one to two hours after perfusion, when the animal is adjusting to its own cardio-pulmonary system, may be a prognostically important time. Several clinical open-heart cases with a low pH postoperatively did poorly.[16] Further work would be necessary to substantiate this viewpoint.

It has been noted that in spite of high flow rates,[2, 15] an accumulation of lactic acid occurs during perfusion, the source of

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*Meeh's formula for surface area = 11.2 x wt. 2/3. If wt. is expressed in kg., answer will be in square decimetres, and to convert it into square metres it is divided by 100.

which is obscure. If anaerobic glycolysis[17] occurred to any extent it could be a possible source. An effort was made in this study to keep the duration between the collection of priming blood and its use in the oxygenator constant and to a minimum. Seligman and his colleagues[18] pointed out that even in severely shocked animals additional parenteral lactic acid is still metabolized. Probably, lactic acid in priming blood is of no real consequence if the blood has not been allowed to incubate either at 37° C. or at room temperature for several hours.

Summary

An alkalosis developed in the five dogs perfused with an extracorporeal circulation at low flow rates (35 ml./kg./minute) and in the five dogs at high flow rates (70 ml./kg./minute); this was considered to be due to the complete elimination of carbon dioxide during the oxygenation process. The alkalosis was evidenced by the high pH values and the low pCO_2 values during the perfusion.

In the five control dogs subjected to thoracotomy, a respiratory alkalosis developed, and it was of the same degree as the alkalosis present during perfusion in the low flow and high flow groups, and was due to the method of artificial respiration during the thoracotomy period.

There was a trend in the low flow group to develop lower pH values than in either the high flow group or thoracotomy control group during the first hour after perfusion. No statistically significant difference occurred in pH values at any interval after perfusion.

The highest pCO_2 values were obtained in the anæsthetized dogs not subjected to thoracotomy and were probably due to the depressed respirations secondary to the anæsthetic effect. The pH fell to levels as low as did the pH in the low flow post-perfusion group or vice versa.

Conclusions

It would seem that, if pure oxygen is used during the oxygenation process in a bubble oxygenator, the development of an alkalosis during the by-pass is a concomitant characteristic. The alkalosis obscured the effects during by-pass that were anticipated as a result of using the arbitrary low and high blood flow rates. Instead, the effects of differential flow rates in this study became apparent in the early post-perfusion period, and then only in a minor degree. It is pointed out that the mean arterial pH values were no lower in the perfusion group of dogs in the post-perfusion period than were the pH values in the control dogs anæsthetized only. Therefore, for the length of by-pass involved, the dogs suffered no severe derangement of acid-base balance in either low or high blood flow groups.

Acknowledgments

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RÉSUMÉ

Ce rapport concerne les changements de pH et pCO_2 survenus chez des chiens qui ont été soumis à la circulation extra-corporelle au cours d'une thoracotomie, en regard de témoins qui n'ont pas été soumis à cette technique. Les auteurs ont aussi étudié l'influence du taux de perfusion sur le pH sanguin de ces animaux pendant l'expérience.

Les chiens furent anesthésiés au penthiobarbital (Pentothal sodique) à la dose de 30 mg./kg. Toutes les déterminations furent faites sur du sang artériel prélevé en deça du cathéter fémoral. L'échantillonnage fut fait avant la perfusion, puis à toutes les cinq minutes durant; puis à partir de cinq minutes après la fermeture de la paroi thoracique.

Les chiens perfusés furent divisés en deux groupes; l'un reçut 35 ml./kg./min. et l'autre 70 ml./kg./min. Les chiens non perfusés furent aussi divisés en deux groupes; dans l'un des groupes, il y eut thoracotomie durant une demi-heure, ce qui correspondait à la durée de la perfusion chez les chiens perfusés; dans l'autre groupe, l'anesthésie dura cinq heures, il n'y eut pas d'opération et les échantillons furent pris à toutes les heures.

Une alcalose se développant durant le circuit extra-corporel, déjà rapportée par d'autres expérimentateurs, provient sans doute de l'usage d'oxygène pur; il est toutefois plus prudent de tolérer un certain degré d'alcalose que de risquer une acidose respiratoire en faisant barboter un mélange d'oxygène et de CO_2 dans le sang durant le processus d'oxygénation.

L'analyse statistique des résultats a montré que: (1) l'alcalose est apparue tant chez les chiens perfusés à un taux lent que chez ceux perfusés plus rapidement, comme résultat de l'élimination complète du CO_2 durant le processus d'oxygénation. Cette alcalose a été démontrée par une élévation du pH et un abaissement du pCO_2 durant la perfusion. (2) Dans les cinq contrôles thoracotomisés, mais non perfusés, une alcalose respiratoire est aussi apparue, qui atteignit le même degré que l'alcalose des chiens perfusés. Elle résultait de la méthode de respiration artificielle employée durant la thoracotomie. (3) Le groupe perfusé à faible taux a eu tendance à montrer un pH plus bas que le groupe à perfusion rapide et que le groupe thoracotomisé mais non perfusé. (4) Les taux de pCO_2 les plus élevés (pH le plus bas) furent obtenus chez les chiens simplement anesthésiés sans opération et furent probablement causés par la dépression respiratoire d'origine anesthésique. Le pH descendit à des niveaux aussi bas que dans le groupe perfusé à faible vitesse.

En conclusion, il semble que si on emploie de l'oxygène pur dans un oxygénateur à bulles, l'alcalose résultante est une caractéristique concomitante durant le circuit extra-corporel. Les effets de la différence du taux de perfusion n'apparaissent qu'une fois la perfusion terminée et sont peu marqués. En effet les valeurs moyennes du pH artériel ne sont pas plus basses après la perfusion dans le groupe de chiens perfusés, que chez les chiens seulement anesthésiés et non perfusés.

Les chiens soumis à la circulation extra-corporelle n'ont pas subi de ce fait de dérangement grave dans leur équilibre acidobasique.

HYPOTHERMIA AND ADRENOCORTICAL RESPONSE TO OPERATION

It is not only the magnitude of trauma but the response of the body to trauma which represents the important factor in recovery. An index of this response may be adduced from the response of the adrenal cortex to trauma or operation, according to MacPhee *et al.* (*Lancet*, 2: 1196, Dec. 6, 1958), who studied urinary corticosteroid excretion, serum and urinary electrolytes and water balance in dogs subjected to laparotomy under hypothermia. They showed conclusively that addition of long-sustained hypothermia to anæsthesia and operation greatly increased urinary corticoid excretion, and presumably steroid production. They suggest that recovery from hypothermia (26-30° C.) increases by three or four times the work done by the adrenal cortex after operation. Hypothermia may insulate the cortex from demands made during trauma, but it does not do so during the later recovery phase.

They also note the difficulty of maintaining a normal alkali reserve during prolonged hypothermia, acidæmia invariably developing, partly respiratory in origin, but also due to loss of fixed base.

CASE REPORTS

DYSPHAGIA DUE TO OSTEOARTHROSIS OF THE CERVICAL SPINE*

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A RETIRED JOCKEY, aged 65, of short stature and with a very short neck, complained to his doctor in January 1958, of hoarseness of his voice and great difficulty in swallowing. He was lethargic and unable to carry out his duties, which were of a sedentary nature. The symptoms had increased and become steadily progressive over a period of four months. Further questioning elicited that he could manage only to swallow soup and milk. All solids stuck "half way down his throat", in fact immediately behind his larynx.

Physical examination.—His general condition was fair. He had pyorrhœa, emphysema, cholelithiasis and a mild hypertension. Radiological examination by Dr. W. G. Hopkirk showed gross cervical spondylosis with a large amount of new bone anterior to the normal line of the bodies from the third to the fifth cervical vertebra. This produced a large bar of bone immediately behind the larynx. The larynx itself showed much calcification of the cartilages. There was also evidence of spondylosis at levels lower than this. A barium swallow showed a moderate degree of pharyngeal obstruction but no evidence of any intrinsic lesion.

Œsophagoscopy was attempted but it was not possible to introduce an œsophagoscope. Examination of the pharynx revealed narrowing but no intrinsic lesion. This narrowing was caused by the anterior protrusion of the aforementioned bar of bone which could easily be distinguished through the posterior pharyngeal wall.

Operation.—Under general anæsthesia supplemented by lidocaine (Xylocaine), hyaluronidase (Hyalase) and adrenaline, with the patient in position as for thyroidectomy, a high wide collar incision was made at the level of the thyroid notch. Skin flaps were raised in the usual way. The sternomastoid muscles on both sides were freed. Access to the cervical spine was obtained by dividing the right omohyoid muscle, retracting the right sternomastoid and carotid sheath to the right and retracting the larynx and pharynx together with the strap muscles to the left. The superior thyroid artery was ligated and divided. Careful dissection resulted in progressive mobilization of the larynx and pharynx. At this point almost the whole of the anterior surface of the third, fourth and fifth cervical vertebræ was exposed to view. The extent of the large bar

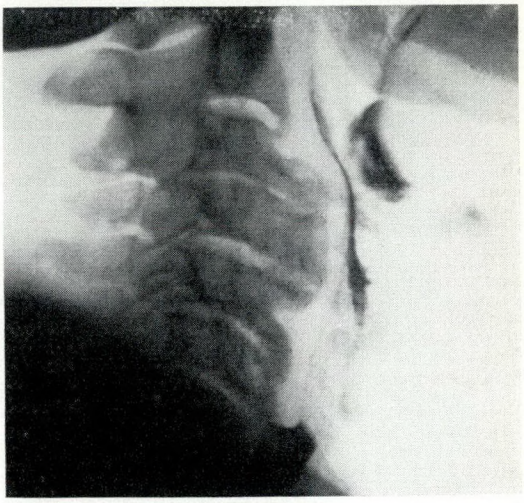

Fig. 1.—Preoperative film, taken at the time of barium examination, showing the massive bone formation on the anterior aspects of the 3rd, 4th and 5th cervical vertebræ. There is no intrinsic lesion in the œsophagus.

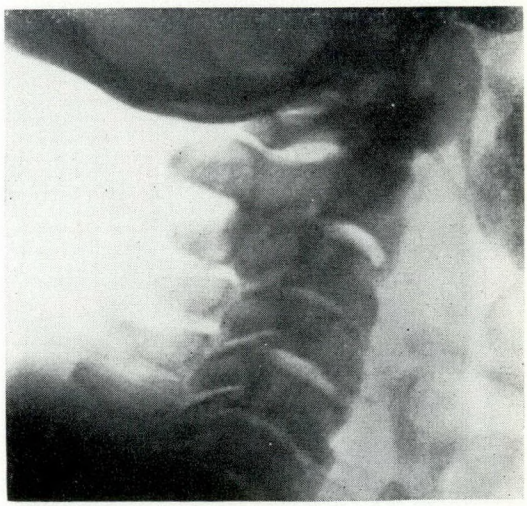

Fig. 2.—Postoperative film. The excess bone has been removed. The patient was able to swallow normally.

*From the King George VI Hospital, Nairobi.

of bone was then clearly defined. It was removed piecemeal with a gouge and mallet. The resultant surface was carefully trimmed with bone nibbling forceps. The wound was closed in the usual way, a small drain being inserted at the angle of the incision on the right.

The postoperative period was uneventful. The patient left hospital 14 days after operation. He was able to swallow all soft foods at this time without difficulty. Two weeks later he could eat any food provided that he chewed it well. This included beef steak. A lateral radiograph of the cervical spine showed that the large amount of osteophytic new bone had been cleanly removed from the anterior face of the third, fourth and fifth cervical vertebral bodies (Fig. 2). The larynx moved up and down normally.

Summary

The interesting features of this case are: (1) That cervical spondylosis (osteoarthrosis) can cause marked dysphagia to the point of inanition. (2) That the anterior surfaces of the cervical vertebral bodies are easily accessible through an anterior collar incision, leading to the plane between the sternomastoid and the strap muscles.

Résumé

Présentation d'un cas: un homme de 65 ans, de petite stature se plaint d'enrouement et de dysphagie. L'examen général révèle de nombreux troubles: pyorrhée, emphysème, cholélithiase et légère hypertension. Un examen radiologique de la nuque montre une forte spondylose cervicale avec apposition d'os nouveau à la face antérieure des corps vertébraux de C3 à C5. Nombreuses calcifications du larynx. L'œsophagoscopie fut impossible, l'appareil ne pouvait être introduit: ceci était dû à une obstruction du pharynx par la masse osseuse apposée en avant des corps vertébraux. L'intervention est décidée: sous anesthésie générale et locale, le patient en position de thyroïdectomie, on pratique une voie d'accès cervicale sur le plan vertébral. Les appositions osseuses sont enlevées au ciseau et au marteau. Les suites post-opératoires furent satisfaisantes, et deux semaines plus tard le patient pouvait s'alimenter normalement.

ENDOBRONCHIAL LIPOMA
PRESENTATION OF A CASE AND REVIEW OF THE LITERATURE*

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Benign mesothelial endobronchial tumours are not common. According to Liebow[1] fibromas, chondromas, leiomyomas, lipomas, lymphomas, myoblastomas, and plasmacytomas do occur. Fibromas and chondromas are the commonest, constituting 80 to 90% of such lesions. Lipomas are stated by Liebow to be the third commonest type of these lesions. The most recent report on endobronchial lipomas was in 1958 by Hutcheson, Ashe and Paulson,[2] who reviewed the scanty literature on the subject, found reports of 18 cases treated surgically and added two of their own. Five further cases were recorded in autopsy reports, bringing the total number of cases recorded to date up to 25. A further case is reported here. It is not proposed to recapitulate the excellent review by Hutcheson, Ashe and Paulson, and the reader is referred to that report.

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Case History

A 51 year old man was referred to the University Hospital, Saskatoon, on February 26, 1958, because of three attacks of right upper lobe pneumonia over the past three years. Each attack occurred during the winter, and was slow to clear. After each attack the patient was slow to regain his strength, but was perfectly fit each summer. Each attack of pneumonia was accompanied by some right-sided pleuritic type pain associated with cough and thick yellow sputum. A series of chest films from St. Peter's Hospital, Melville, Saskatchewan, showed that the patient had had two bouts of consolidation in the right upper lobe in the past, but a chest film on January 3, 1957, was clear and no mediastinal shadow

was present at that time. At no time had he lost weight and indeed he was somewhat obese. He was a smoker.

On physical examination, the vital signs were normal and no abnormal signs were found on chest examination. The hæmoglobin value was 15.2 grams per 100 ml. and the erythrocyte sedimentation rate was 40 mm. in one hour.

X-ray films of the chest showed a density adjacent to, and fused with, the right upper mediastinum above the level of the hilum of the lung (Fig. 1). At fluoroscopy this mass was thought to pulsate, and some observers felt that this represented an aneurysm of the ascending aorta. However, the authors did not agree with this interpretation because the shadow spread upward above the level of the aortic arch, which was clearly seen. Also, there were no cardiological signs to fit in with an aneurysm of the ascending aorta, and the serological tests for syphilis were negative; furthermore, one year previously no such mass was present and an aneurysm would scarcely develop in so short a time. It was thought that an atelectasis of the right upper lobe of the lung was the most likely explanation of the mass.

Oblique planigrams failed to solve the problem because they did not cut across the right upper lobe bronchus. A bronchoscopy showed the right upper lobe bronchus to be blocked one centimetre from its origin by a rounded swelling projecting from the upper and posterior wall. This swelling was smooth, not ulcerated, and resembled a lipoma. Biopsy was not possible because of the situation of the mass. Bronchograms confirmed a complete block of the right upper lobe bronchus (Fig. 2).

Thoracotomy by one of us (E.M.N.) revealed a complete atelectasis of the right upper lobe with large infected lymph nodes around the upper hilum. A right upper lobectomy was performed. The patient made a good recovery.

Pathological Examination (Dr. D. F. Moore)

The specimen consisted of an atelectatic right upper lobe weighing 185 grams. Right at the resection line was a broad based, sessile, soft yellow tumour measuring 1.3 x 1.0 x 1.0 cm. Its outer aspect was smooth and on section (Fig. 3) it appeared to consist of bright yellow fat. It had produced complete occlusion of the bronchus with resultant gross bronchiectasis.

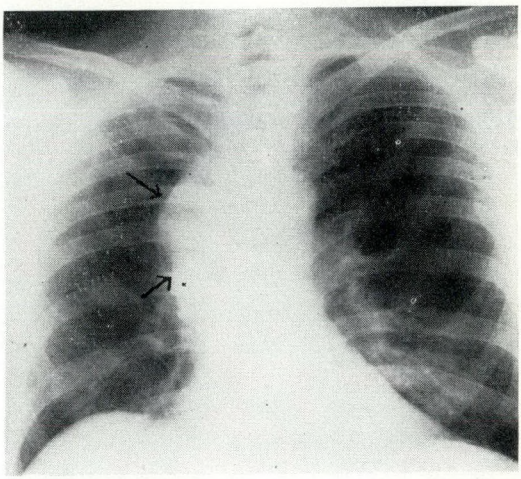

Fig. 1.—Radiograph showing collapsed right upper lobe of lung (see arrows).

Microscopic Examination

Sections of the tumour showed it to be a lipoma composed of fatty tissue in which were embedded a few mucus secreting glands. A portion of the tumour lay deep to the submucosa of the bronchial wall. A portion lay within the lumen of the bronchus, partially covered with columnar epithelium lightly infiltrated with chronic inflammatory cells.

COMMENT

At first thought, this patient's history would suggest a bronchogenic carcinoma. But on the other hand, the history was of

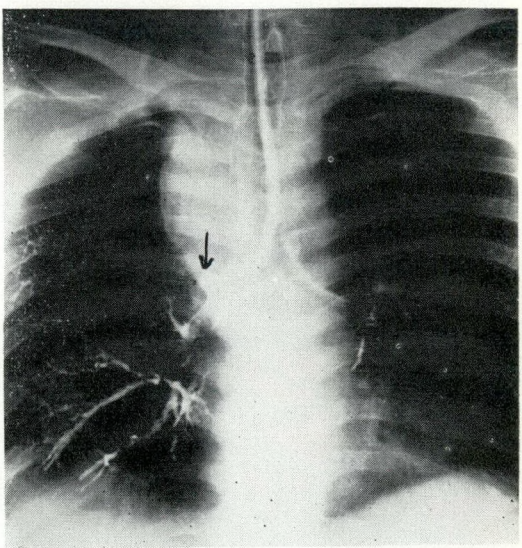

Fig. 2.—Bronchogram showing complete stenosis of right upper lobe bronchus (see arrow).

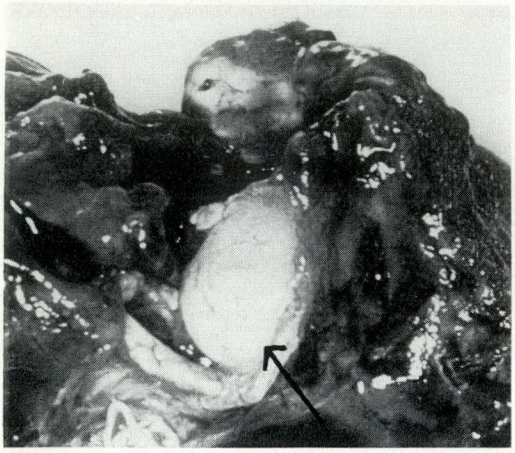

Fig. 3.—Photograph of right upper lobe of lung showing the endobronchial lipoma completely blocking the bronchus (see arrow).

three years' duration, and the patient was still well. He was known to have had a clear chest radiograph one year previously. It was therefore felt that this man must have some peculiar endobronchial lesion blocking the bronchus and producing atelectasis. An adenoma would be the most likely lesion to do this, but there was no history of hæmoptysis and at bronchoscopy the mass blocking the bronchus was smooth, non-vascular, and yellowish in colour. It was therefore thought that there was a good chance that this would be a benign lesion.

The diagnosis was also rendered difficult by the location of the tumour in the right upper lobe bronchus; it would have been even more difficult if the tumour had been in the left upper lobe bronchus. Even with the retrograde telescope, it was hard to get a clear view of the lesion and impossible to biopsy it. The other point worth commenting upon is the way an atelectatic upper lobe will simulate a mediastinal lesion.

Another aspect worthy of comment is the malign effect of any chronic endobronchial obstruction, whether it be foreign body, stricture, or benign tumour. Such lesions, though small in size, will destroy large areas of pulmonary tissue by causing recurrent infections resulting in abscess formation, bronchiectasis and fibrosis. A full early investigation using planigrams, bronchoscopy and bronchograms, is therefore necessary. Lipomata arise from the submucosal areolar tissue. Ideally, therefore, they should be removed by endoscopic methods, or if this is not possible by bronchotomy. But for these methods to be successful, the lung tissue distal to them should have suffered no irreparable damage.

Summary

The world literature contains records of 25 endobronchial lipomata. A 26th case is hereby added.

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Résumé

Présentation du cas d'un homme de 51 ans hospitalisé à la suite de trois épisodes pneumoniques du lobe supérieur droit, qui étaient chacun très longs à guérir et s'accompagnaient d'une très forte fatigue générale. Les radiographies montraient une opacification, continue avec le médiastin supérieur droit. On pensa à un anévrysme. Mais cette hypothèse ne fut pas retenue; il n'y avait aucun signe cardiaque et les examens sérologiques pour la syphilis étaient négatifs.

Une bronchoscopie montra la bronche lobaire supérieure droite bloquée à un centimètre de son origine par une masse ronde venant d'en haut et en arrière, ressemblant à un lipome. Une thoracotomie fut pratiquée au cours de laquelle on procéda à une lobectomie supérieure droite, ce lobe étant totalement atelectasié. Les suites furent sans histoire. L'examen anatomopathologique confirma le diagnostic de lipome.

L'intérêt de ce cas réside dans la rareté de cette lésion et dans l'établissement du diagnostic différentiel.

ANGIOBLASTIC SARCOMA IN POST-MASTECTOMY LYMPHŒDEMA

W. L. OGILVY, M.B., Ch.B., M.Sc.,* R. H. FRANKLIN, M.B., B.S., F.R.C.S.† and IAN AIRD, Ch.M., F.R.C.S.,‡ *Montreal* and *London, Eng.*

THIS CASE is reported in an attempt to re-emphasize the necessity of long term follow-up of patients apparently cured of carcinoma of the breast by radical mastectomy.

CASE REPORT

In February 1951, at the age of 49 years, Mrs. M.L. had a left radical mastectomy performed. The lump in the upper outer quadrant of the breast had been present for one month, and there was no clinical evidence of axillary lymph node involvement. Histologically, the breast lesion was a spheroidal cell carcinoma and one axillary node was replaced by tumour. After operation she received deep x-ray therapy.

Swelling of the left arm was first noted at a routine follow-up visit six months after the operation. Continued follow-up revealed no evidence of local recurrence or distant metastases, but when seen in February 1958, the patient complained of a small reddish-blue nodule on the anterior aspect of the upper arm at the lateral end of the previous mastectomy scar. The lump had been present for two months and she thought it was rather odd that she should have a "bruise" with no associated history of injury. She had also noticed a marked increase in size in the swollen arm over the previous two months.

Figs. 1 and 2 show the situation of the lesion and the marked lymphœdema of the arm. The lesion was 2 cm. in diameter and raised 1 cm. above the surface. It had a reddish-blue marbled appearance and felt firmly cystic. There was a surrounding halo which resembled a subsiding bruise in colour. A diagnosis was made of angiosarcoma developing in a post-mastectomy lymphœdematous arm.

On February 7, 1958, excisional biopsy was attempted but on incising the skin it was apparent that the lesion was much deeper and had spread much further around the arm than had appeared clinically. In this respect, the similarity between the lesion and an iceberg was very marked. The vascularity of the lesion was amply confirmed.

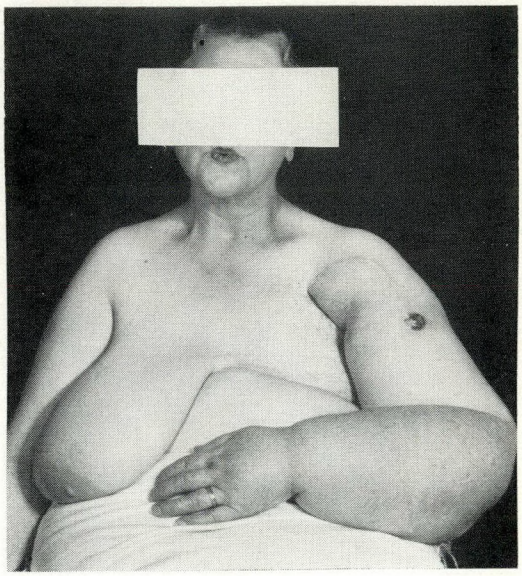

Fig. 1.

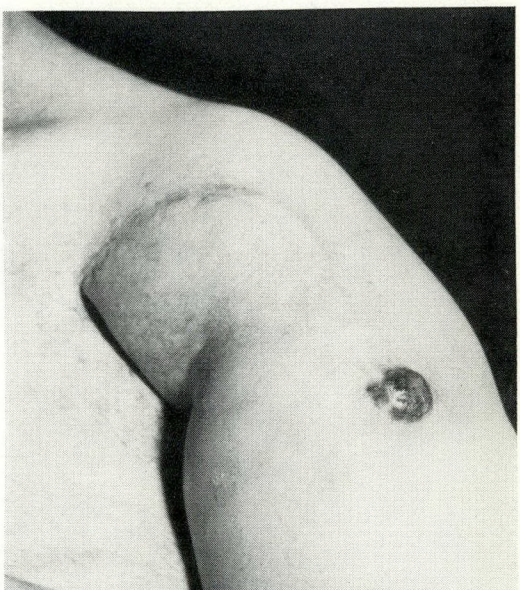

Fig. 2.

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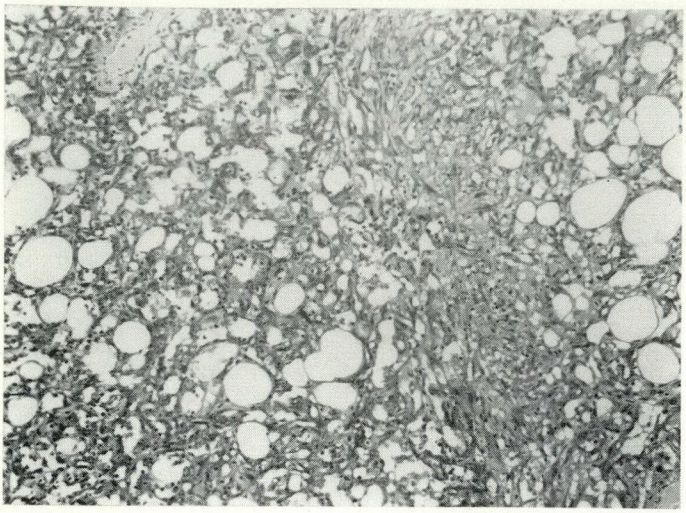

Fig. 3.—Section to show proliferation of blood vessels through fatty tissue. The papillary nature is shown clearly in the reticulum impregnated section (x 104).

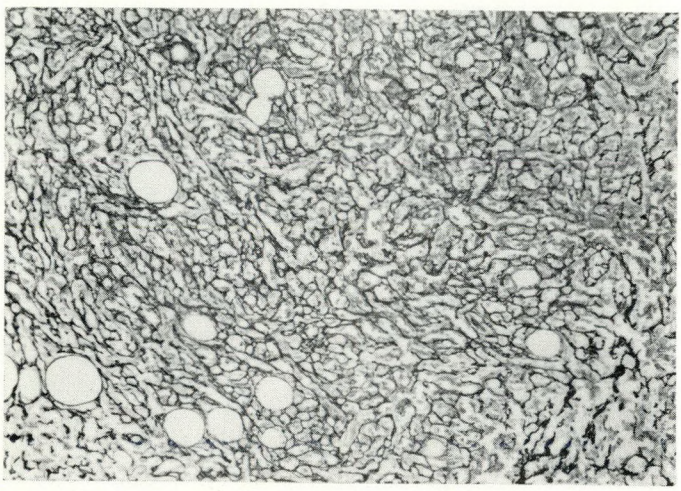

Fig. 4.—Reticulum impregnated section showing proliferation of vessels through fatty tissue (x 104).

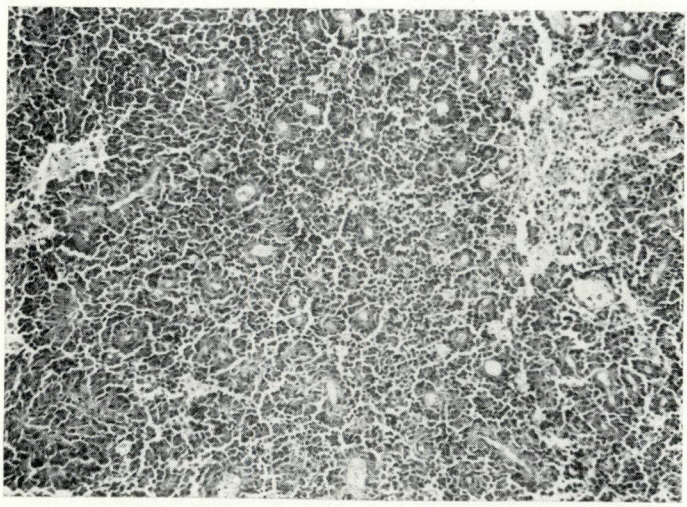

Fig. 5.—Section to show poorly differentiated tumour area (x 104).

The malignant nature of the lesion was confirmed histologically, and a left interscapulothoracic amputation was performed on February 18, 1958. Her immediate postoperative course was very satisfactory, but a small recurrent lesion was noted in the scar 10 weeks after the fore-quarter amputation. This lesion was excised, and was shown histologically to be similar to the initial tumour. Chest radiography revealed no pulmonary lesion.

Pathology

The initial biopsy specimen sections showed a malignant connective tissue tumour infiltrating the whole thickness of the dermis and subcutaneous fatty tissue. The most prominent pattern was one of elongated, cleft-like, sinuous spaces, ramifying and anastomosing in the fibro-fatty tissue to form a plexiform network of fine tubular structures. These spaces were lined completely or incompletely by hyperplastic angioblasts, of spindle or ovoid shape, which often protruded into the vascular lumina they lined; they had relatively large, hyperchromatic nuclei and prominent nucleoli; occasional mitoses were seen in these endothelial cells; sometimes the proliferating angioblasts virtually filled the vascular lumen producing solid cords. No sharp separation could be drawn between hyperplastic and neoplastic vessels. Many of these vascular lumina contained red blood cells but others were empty.

Other areas of the tumour consisted of solid sheets of undifferentiated cells with numerous mitoses; the nature of the tumour was quite obscured in such areas. Some parts had a curiously papillary pattern, mantles of tumour cells surrounding a vascular core lined by flat endothelial cells; the vessels in these papillary patterns probably represented only perivascular survival of tumour. There was abundant hæmorrhage into fibrous stroma and fatty tissue rendering difficult the interpretation of the presence of blood in the well formed neoplastic channels. Small venules were invaded by tumour cells; staining for elastic tissue confirmed this.

Although Stewart and Treves[9] called this type of lesion a lymphangiosarcoma, in this particular instance the origin from blood vessels or lymphatics remains in doubt.

The pathological examination of the specimen after fore-quarter amputation revealed no additional conclusive evidence that would incriminate blood vessels as opposed to lymphatic vessels as the source of origin of the tumour.

Figs. 3, 4 and 5 are photomicrographs of typical sections of the tumour.

Discussion

That a new menace has arisen to plague the long term survivor of radical mastectomy is probably not true. This tumour was first recognized in 1948 by Stewart and Treves[9] when they reported six cases, and further case reports have appeared in the literature since then.[2-8] Undoubtedly cases were seen before 1948 and presumably misdiagnosed as recurrent carcinoma or Kaposi's sarcoma.

The clinical similarity in all reported cases is very striking. A radical mastectomy is performed and is followed immediately, or within the first year, by lymphœdema of the arm. Thereafter, there is a long latent interval of several years—12 years average in the reported cases to date—followed by the development of this highly malignant tumour in the lymphœdematous arm.

Clinically the lesion does not look like recurrent carcinoma, although on rare occasions it might be confused with a colloid carcinoma. Grossly, the lesion may also be taken as an example of Kaposi's sarcoma, but the two differ markedly in their development and clinical behaviour. It is true that cases of Kaposi's sarcoma are recorded in which the initial lesion is œdema of the affected extremity. In cases after mastectomy, however, the œdema clearly precedes an angiosarcoma, which must in some way be secondary to that œdema. It would be too notable a coincidence for Kaposi's sarcoma to occur so often independently in a patient after mastectomy and always in the arm on the affected side.

Two distinct diseases are apparently involved: (1) Kaposi's disease, an angiosarcoma developing in an extremity previously normal; (2) a separate tumour, developing in a limb already the seat of chronic œdema, either after mastectomy or spontaneously. Histologically also, confusion may arise with Kaposi's sarcoma, especially if only a very small biopsy of the lesion is taken. Some of the cases of Stewart and Treves[9] were so labelled initially, but on

review proved to be examples of lymphangiosarcoma. These authors give a very full account of the lesion as a pathological study.

Why should such a tumour develop at all? This question has led to a lot of speculation, but the reason for the development of lymphœdema in the arm is also obscure. The only common factor in every case reported was the development of a swollen arm immediately or soon after radical mastectomy. In one of the early cases[9] the presence of carcinoma in the breast is not proven, so it must be supposed that the operation was performed for a benign lesion. The lesions on the arm have occurred with or without evidence of previous lymph node involvement, with or without previous wound infection, with or without preoperative or postoperative radiotherapy, at a wide variety of ages and after latent intervals varying from six to 24 years. Stewart and Treves postulated the presence of a systemic carcinogen not only as a cause of the initial mammary cancer, although in one of their cases the presence of the initial mammary cancer is unsubstantiated. They support this hypothesis by noting that no similar tumour has ever been described in other chronic lymphœdematous conditions, e.g. filariasis. More recently, however, Aird, Weinbren and Walter[1] reported a case of angiosarcoma which developed in a lower limb which was the seat of spontaneous lymphœdema. The diagnosis of spontaneous lymphœdema was well substantiated. The patient had never been operated on previously, and was free from other malignant disease. This case makes the thesis of a general systemic carcinogen less tenable.

The origin of the tumour in the present report is unknown. There is vessel formation, some containing red blood cells and some empty, so is this a blood vessel tumour or a lymphatic vessel tumour? Stewart and Treves called it a lymphangiosarcoma, and other authors reporting since then have accepted this name although some express doubt about it. Cruse, Fisher and Usher[2] state that the biopsy specimen in their case contained so much altered blood that it was confused with a melanoma until the pigment was identified by stains for free iron. Jessner, Zak and Rein[6] consider that the lesion must consist of a mixture of lymphatics and blood vessels rather than being purely lymphangioblastic. Hilfinger and Eberle[5] are not totally convinced that this tumour arises solely from lymphatics.

Irrespective of the source of origin, the tumour clinically is extremely malignant. Early recognition of the lesion and ablation of the extremity by fore-quarter amputation appears to be the best line of attack, but the prognosis in the cases reported to date can be measured in months rather than years. These tumours are considered to be radio-resistant, but Rawson and Frank[8] believe that the earlier lesions reported as receiving radiotherapy had insufficient dosage, and they reported one patient who was well with no evidence of local recurrence or distant metastases one year after radical radiotherapy. Their method is to give 3500 to 4000 r in a three week period; they agree that recurrence will be inevitable with smaller doses. Thus some of these tumours should be classified as radio-sensitive, but sensitivity to radiation is lowered with recurrence.

It appears highly unlikely that lymphœdema per se is a premalignant condition; some other unknown factor must be postulated. With the present surgical approach to the treatment of mammary cancer and the efficient radiotherapy these patients receive, the high percentage of 5-year and 10-year survivals is most encouraging, but a large percentage of women after radical mastectomy develop lymphœdema of the arm to a greater or lesser degree. The long term survivor with concomitant lymphœdema is the candidate for the development of an angioblastic sarcoma in the swollen arm.

Summary

A case is reported of angioblastic sarcoma developing in a post-mastectomy lymphœdematous arm just under seven years after radical mastectomy for carcinoma of the breast. The exact site of origin of the tumour from blood vessels or lymphatics could not be determined in this instance.

This type of neoplasm was recognized first in 1948, and a brief review of the

reported cases since then is included. The development of the lesion as a true clinical entity, as seen by the similarity of all the case histories, is noted—radical mastectomy, lymphœdema of the arm, latent interval of several years, then the development of the tumour in the swollen arm.

Interscapulo-thoracic amputation is the treatment of choice, the tumour being considered radio-resistant.

The prognosis is poor.

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Résumé

Ce cas est décrit pour rappeler la nécessité de suivre pendant longtemps les patientes apparemment guéries d'un cancer du sein par mastectomie.

La malade fut opérée (ablation radicale du sein gauche) à l'âge de 49 ans, et reçut de la radiothérapie. Lors d'un examen de routine six mois plus tard, on nota une enflure du bras gauche. Sept ans plus tard la patiente se plaint d'un petit nodule bleu violacé apparu sur la partie externe de la cicatrice de mastectomie: de plus l'enflure du bras a augmenté. Après avoir pratiqué une biopsie, on pose le diagnostic d'angiosarcome: il est procédé à une amputation interscapulo-thoracique. Les suites post-opératoires furent satisfaisantes, mais une récidive se fit dix semaines plus tard.

On trouve peu de ces cas dans la littérature. Mais il semble qu'assez souvent le diagnostic n'a pas été fait et que l'on a simplement cru à une récidive du néoplasme. Le diagnostic différentiel avec un sarcome de Kaposi se pose également. Pourquoi un angiosarcome apparaît-il ainsi après un cancer du sein? Ceci n'est pas encore expliqué. Mais le développement de cette lésion constitue réellement une entité clinique distincte, étant donné les ressemblances que présentent les cas publiés. Le pronostic est mauvais.

DISEASES OF THE THYROID AND PARATHYROID GLANDS. B. J. Ficarra. 295 pp. Illust. Intercontinental Medical Book Corporation, New York, 1958. $8.50.

Bernard J. Ficarra's "Diseases of the Thyroid and Parathyroid Glands" is a fine book that fills a need for everyone interested in surgery.

As the author states in his introduction, the book was written for the general surgeon. A very good and comprehensive synthesis is provided in the first chapters on the anatomy, physiology and biochemistry of the thyroid gland. This is followed by an evaluation of diagnostic methods with special emphasis on the value of the 24 hour I^{131} uptake.

In the chapter on endemic goitre, the author discusses the geographical areas which are iodine free and the role of calcium as a goitrogenic agent.

Chapter 5, on mental symptoms in hyperthyroidism, is very important and gives the psychosomatic aspect of the disease its true place within the syndrome. The discussion of hyperthyroidism during puberty, pregnancy and the climacteric illustrates the relationship between the thyroid gland and the reproductive system.

The descriptions of hyperthyroidism and its management and of cancers of the thyroid and their treatment give the general surgeon good criteria of what to do and what not to do in his practice.

The last chapter deals with the parathyroid glands, their pathology and treatment.

In conclusion, it is felt that this is a fine book which should be read and/or consulted by all who treat patients for diseases of the thyroid and parathyroid glands.

SEPTUM OF THE AQUEDUCT OF SYLVIUS: A DEVELOPMENTAL ANOMALY*

R. G. LECKEY, M.D. and T. P. MORLEY, F.R.C.S., *Toronto*

A MEMBRANE occluding the aqueduct of Sylvius is a rare congenital anomaly and only a few cases have been reported.

CASE REPORT

C.B., a 23 year old man, was admitted to the neurosurgical service of the Toronto General Hospital on October 26, 1955. Three years before admission he had had a steady frontal headache which persisted for two weeks and then cleared spontaneously. This headache recurred the following year and similarly subsided. Four weeks before the present admission the headache returned but it occurred in episodes and was followed within a week of its onset by nausea and vomiting. The headache was aggravated by movement but the patient obtained some relief by steadying his head with his hands. Between the episodes of headache, nausea and vomiting his appetite was good and he had no complaints. During this period, however, he lost 20 lb.

There was no previous history of significant illness. He had been a semi-skilled labourer leading a normal life.

On physical examination, apart from the pain of the headache which was increased by movement, there were no abnormalities. There were no abnormal neurological findings, no enlargement of the head and no papillœdema.

Investigations.—(1) Routine skull films were normal. (2) The electroencephalogram did not show any significant abnormality. (3) Lumbar cerebrospinal fluid (CSF) pressure was 200 mm. and the fluid had a normal cell count and a protein content of 19 mg. per 100 ml. (4) A lumbar air encephalogram failed to demonstrate the ventricles, the air passing only to the subarachnoid space. (5) Air ventriculograms (occipital burr holes) showed a dilated ventricular system (Fig. 1). No intracranial mass was outlined. It appeared that the fourth ventricle had taken part in the general dilatation and that the obstruction to the passage of air was at the outlets of the fourth ventricle, an interpretation of the films that was later shown to be wrong. (6) The protein content of the ventricular fluid was 7 mg. per 100 ml.

Operation.—Exploration of the posterior fossa with the patient sitting showed that both cerebellar tonsils were herniated to the upper border of the arch of the atlas. The cerebellar hemispheres were normal in appearance and texture. There was no evidence of an Arnold-Chiari malformation or of any vascular abnormality. The pia arachnoid of the cisterna magna looked normal and there was no sign of old inflammatory thickening. The lower part of the floor of the fourth ventricle was normal in appearance but no CSF passed through the ventricle when the jugular veins were compressed. When the tonsils were retracted apart a shining, diaphanous membrane at the lower end of the aqueduct was seen bulging down into the fourth ventricle (Fig. 2) and when it was incised CSF gushed out. Saline could then be irrigated from the lateral ventricle into the fourth ventricle without obstruction. The opening in the membrane was enlarged so that it fell away on all sides to disclose a large chamber formed by the dilatation of the aqueduct (Fig. 3). The fourth ventricle floor with its anatomical landmarks could be discerned below and separate from the dilated aqueduct, the boundary between the two being marked by the attachment of the membrane to the lower end of the aqueduct wall. It is important that this anatomical point should be emphasized, because in the lateral view of the ventriculogram air can be seen in the cul-de-sac of the aqueduct in a position normally occupied by the fourth ventricle. In a sense this air shadow did mark the position of the ventricle, but the air was contained by the membrane which bulged down into the ventricle (Fig. 4).

Progress.—The patient was discharged two weeks after operation, and during the subsequent three years has been free from symptoms. Before his discharge residual air in the lateral ventricles was radiologically demonstrated passing freely through the fourth ventricle and out into the subarachnoid space. His completely normal health since his operation has been taken to prove that the aqueduct has remained patent.

DISCUSSION

Nothing was found in the investigation of this patient or at operation to suggest an inflammatory or neoplastic cause for the

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obstruction of the aqueduct, although no biopsy was made of the frail membrane. There was no previous history of inflammatory disease and the appearance of the fourth ventricle, aqueduct and cisterna magna did not suggest it. The excellent recovery the patient made postoperatively and his subsequent freedom from symptoms are sufficient evidence that the membrane was the cause of the internal hydrocephalus.

Russell[1] classified developmental obstruction of the aqueduct of Sylvius into three groups. (1) Stenosis, when the aqueduct is histologically normal but abnormally small. (2) Forking, with two distinct channels in the midsagittal plane separated by normal nervous tissue. (3) Septum formation; Russell described two cases of her own and referred to three others in the literature. One of her patients lived to be at least 20 but was mentally retarded, developed profound neurological changes and became bedridden, while the other died when eight years old with gross hydrocephalus and neurological disorders. Orton[2] described a case of hydrocephalus due to a septum in the aqueduct. This patient lived 46 years but was of poor mentality and had obvious hydrocephalus. Versé[3] presented the case of a patient with hydrocephalus who died at eight and three-quarter years and in whom the microscopic examination of the necropsy material demonstrated small ependymal canaliculi by-passing the membrane. Rowbotham[4] reported an 11 year old girl with a septum which had become perforated; close to it was a saccular aneurysm projecting into the lumen of the aqueduct.

Since these cases (all referred to in Russell's work[1]) were recorded there has been, as far as we are aware, only one report of a septum similar to that described here. Petit-Dutaillis *et al.*[5] record that a 35 year old man (Observation XVIII) with minimal neurological findings had an obstruction of the aqueduct. The posterior cranial fossa was explored and a membrane at the lower extremity of the aqueduct was opened. Five years later, the patient still had some of the original associated neurological deficiencies but he was able to do light work and was free from the headache

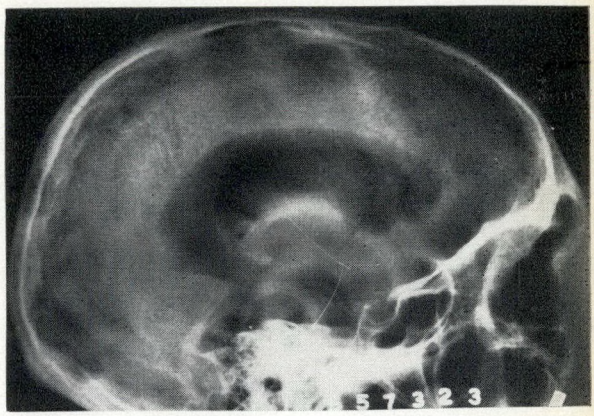

Fig. 1.—Ventriculogram. An air shadow is seen in the position of the fourth ventricle and there is a moderate internal hydrocephalus.

of which he had once complained. In addition, Horsey and Van Patter[6] demonstrated a thick membrane composed of glial tissue that had, they believed, been laid down across the aqueduct following hæmorrhage from a congenital arteriovenous anomaly of the midbrain.

The chief interest of these cases, however, lies not in the morbid anatomy of the anomaly but in the hydrodynamics of the CSF circulation in the presence of aqueduct obstruction. For example, the age of onset of symptoms in obstruction due to congenital anomalies may be delayed and the need for treatment may not arise until early adult life. This is puzzling because, presumably, the gross anatomy of the anomaly does not change between the time it is laid down in the fetus and the time it gives rise to trouble years later. Why then, as was the case in this patient, should 20 years pass before signs of aqueduct obstruction develop? Beckett, Netsky and Zimmerman[7] demonstrated two specimens where the CSF, dammed up by an obstruction of the aqueduct, had escaped in one case through the corpus callosum which had in consequence a demonstrable defect in it, and in the other by what they believed to be permeation through the thinned-out wall of a posterior third ventricle hernia. They point out that other reports have been made of CSF escaping in the same way from the wall of the lateral ventricle. They suggest also that the production of CSF may be suppressed in the presence of chronic ob-

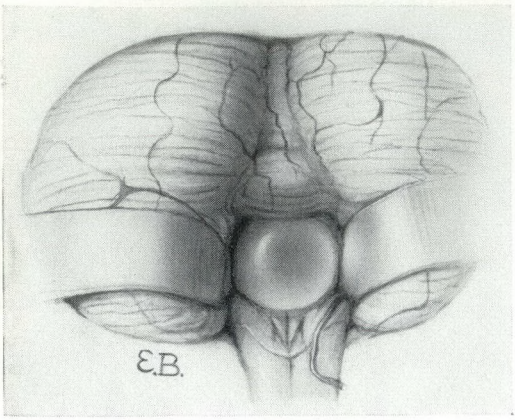

Fig. 2.—Appearance of the membrane at operation. Note that the floor of the fourth ventricle can be seen, which confirms that the attachment of the membrane is above the cavity of the ventricle.

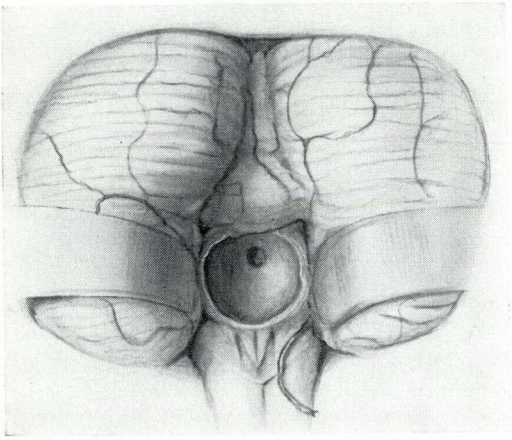

Fig. 3.—After incision of the membrane; there is an enormous dilatation of the lower part of the aqueduct.

struction. Woollam and Miller[8] after examining the microscopical anatomy of stenosed aqueducts were unable to explain why hydrocephalus developed in some instances and not in others in spite of the similarity in size of the aqueducts. Sweet *et al.*[9] and Bakay,[10] using radioactive tracers to study the formation and circulation of the CSF, showed that in addition to the choroid plexus all the surfaces of the CSF pathways allow many constituents of the CSF to enter and leave through them. But that is not to say that the fluid is formed at those surfaces or that there is a *flow* of fluid across them in either direction. Nor is there evidence that fluid can be absorbed in substantial quantities from these surfaces if it is cut off from its normal route of departure. Indeed, the clinical evidence points overwhelmingly to the contrary, and the traditional concepts of CSF formation and circulation cannot thus be dislodged. This point has also been made by Selverstone[11] and by Bering.[12] Tracer studies do, nevertheless, provide useful information on ionic interchange between blood, brain and CSF.

It would simplify the explanation of the delay in obstructive symptoms in this case if the ability to absorb fluid could be ascribed to the ventricle walls. Even if this were possible in only small amounts —which is plausible—it could have occurred at a sufficient rate to keep pace with production until some unknown factor interposed to upset the precarious balance. In that case, however, retention of protein in the ventricular CSF would have been expected (Sweet and Locksley[13] have shown that the main site of protein absorption from the CSF is at the arachnoid villi) but analysis of the fluid showed that its protein content was normal. On this hypothesis, too, it would be desirable to postulate at the same time a suppression or reduction of CSF production from the choroid plexus. This may have been a factor contributing to the evident physiological adjustment that

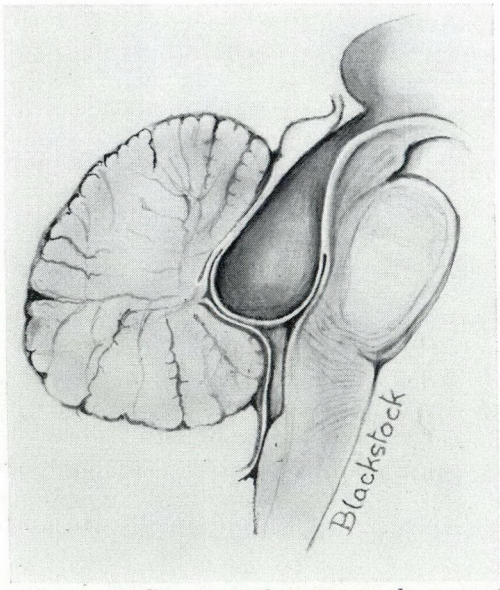

Fig. 4.—Midline sagittal section to demonstrate the membrane. Although attached to the lower end of the aqueduct wall, it ballooned into the fourth ventricle.

the brain had so successfully made in the face of aqueduct obstruction and there is, in fact, some experimental support for this view. Sweet *et al.*[9] measured the volume of fluid that had to be withdrawn from the lateral and third ventricles in order to keep the pressure within them constant; when the ventricles were normal in size the volume of fluid withdrawn was 40 to 100 ml. a day, but in gross obstructive hydrocephalus it was 15 ml. a day. Yet perhaps this is not the whole explanation. There remains the consideration of the occluding membrane itself. Alteration, with the passage of time, in its cytological and physical properties would be expected and this would explain the long delay before obstruction to the flow of CSF developed; before this change in quality of the membrane took place the CSF passed through it fast enough to prevent the occurrence of hydrocephalus.

Summary

A case is described of a congenital septum of the Sylvian aqueduct in which CSF obstruction did not arise until early adult life. Simple incision of the membrane has been followed by complete and lasting (three years) relief of symptoms and signs of CSF obstruction. Possible explanations are offered to account for this delay in onset of obstruction until the age of twenty.

Acknowledgment

The authors thank Miss E. Blackstock of the Department of Art as Applied to Medicine, University of Toronto, for the illustrations.

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Résumé

L'obstruction de l'aqueduc de Sylvius par une membrane est une malformation congénitale rare. Les auteurs présentent le cas suivant:

Un homme de 23 ans est admis au service de neurologie de l'Hôpital général de Toronto pour des céphalées violentes durant depuis trois ans; aucun autre symptôme ou phénomène pathologique. A l'examen, on ne trouve rien à signaler sauf une certaine hydrocéphalie. Diverses épreuves furent pratiquées, parmi lesquelles: un encéphalogramme gazeux par voie lombaire qui ne montra pas les ventricules; et une ventriculographie gazeuse transoccipitale qui mit en évidence des ventricules dilatés, y compris le quatrième. On procéda alors à une exploration de la fosse postérieure; il n'existait aucune lésion inflammatoire mais une hernie des amygdales; le plancher du quatrième ventricule était d'aspect tout à fait normal, mais la compression des jugulaires ne provoquait aucun écoulement de liquide céphalo-rachidien. La partie terminale basse de l'aqueduc était obstruée par une fine membrane diaphane qui fut largement excisée. Les suites post-opératoires furent sans incident et le malade quitta le service deux semaines plus tard. Depuis, soit main-

tenant trois ans après cette intervention, il jouit d'une excellente santé.

Les auteurs passent ensuite en revue les cas de ce genre qu'ils ont pu trouver dans la littérature. Dans le cas présent, il est particulièrement surprenant de constater que, chez ce patient, la circulation du liquide céphalo-rachidien s'est effectuée sans trouble pendant environ vingt ans, puisque ce n'est qu'à cet âge que les premiers symptômes sont survenus. Trois suppositions peuvent tenter d'expliquer ce fait: (1) des échanges furent possibles à travers cette membrane pendant assez longtemps, par une sorte d'osmose: ils furent bloqués à la suite d'une modification histologique ou d'un changement de perméabilité (2) une certaine absorption se faisait au niveau des parois mêmes du ventricule (3) enfin, la pression qui provoqua l'hydrocéphalie fut peut-être suffisante pour diminuer la sécrétion du liquide céphalo-rachidien.

CONGENITAL ABSENCE OF ULNA*

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THIS REPORT describes a case of congenital absence of the ulna in a child admitted to the Hospital for Sick Children, Toronto. Congenital absence of the ulna is the second rarest type of congenital absence of a long bone, the rarest type being an absence of humerus. Congenital absence of humerus (other than associated with congenital amputation of the arm) is associated with an absent radius, but has not been described with an absence of the ulna.

Rabaud and Havelocque[10] (1924) noted 71 cases of congenital absence of ulna in the literature. Few articles on this subject have since been submitted. The diagnosis is probably often missed, as Birch-Jensen[1] (1949) noted this congenital defect once for 200,000 births.

CAUSE

Isolated examples of a strong hereditary factor have been reported;[7] Roberts[11] (1898) noted the deformity in three successive generations; most cases, however, have no positive family history.

The exact contributions of endogenous and exogenous factors to the development of congenital anomalies is well discussed elsewhere[2] and is beyond the scope of this report.

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DESCRIPTION

The anomaly is most often unilateral, and commoner on the right side. This predominance of right sided lesions has been previously noted with congenital absence of other long bones (Farmer and Laurin, 1957).

The presenting deformities will vary; nonetheless, there is a remarkably constant pattern. Kümmel[13] (1895) distinguished three types of congenital absence of ulna on the basis of varying radio-humeral relationship.

Type 1.—Normal radio-humeral joint; the radius is intact or slightly curved.

Type 2.—Bony ankylosis of radius and humerus; if a proximal segment of ulna is present, the latter is fused to the radius and/or to the humerus.

Type 3.—Proximal dislocation of the radius.

In all three types, the absence of ulna may be complete or incomplete, but the associated hand anomaly is worse in Types 2 and 3.

The absence of ulna is more often incomplete, as noted by Klippel[6] (1925) and by Piulachs[9], the distal end being missing more frequently as in cases of congenital absence of the radius. In such instances ossification is always delayed, and early radiographic examination may give the false impression of a complete absence of ulna.

In the normal limb, the ulna is responsible for most of the stability at the

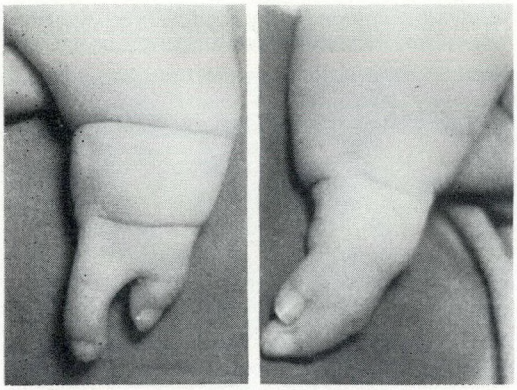

Fig. 1.—Deformity at six weeks of age: shortening, adactyly and syndactyly.

elbow joint, while the radius offers the widest articulating surface of the wrist joint. For that reason, patients with congenital absence of the ulna or of the radius have the major deformity at the elbow or wrist respectively. In cases of congenital absence of the ulna, the deviation at the wrist is rarely severe but the elbow joint is usually abnormal.

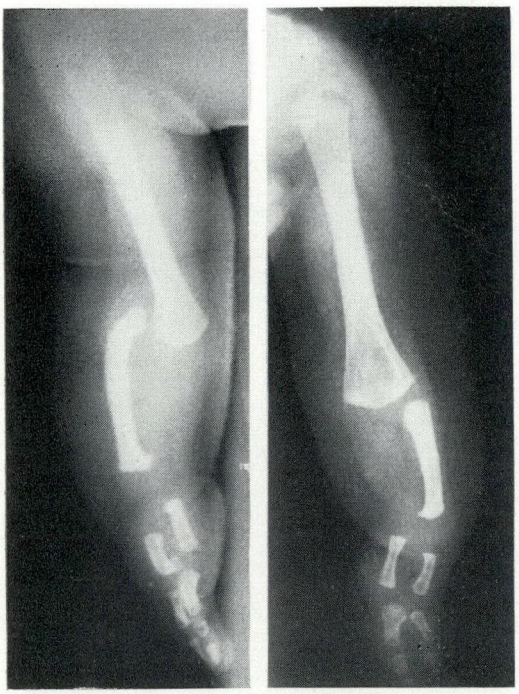

Fig. 2.—Radiographs at six weeks of age: Bilateral absence of ulna may be confused with congenital absence of radius. Radiographic pictures show: (1) Bilateral complete absence of ulna; (2) Proximal dislocation of head of radius (right); (3) Radiohumeral synostosis (left); (4) Defect on the ulnar side of both hands.

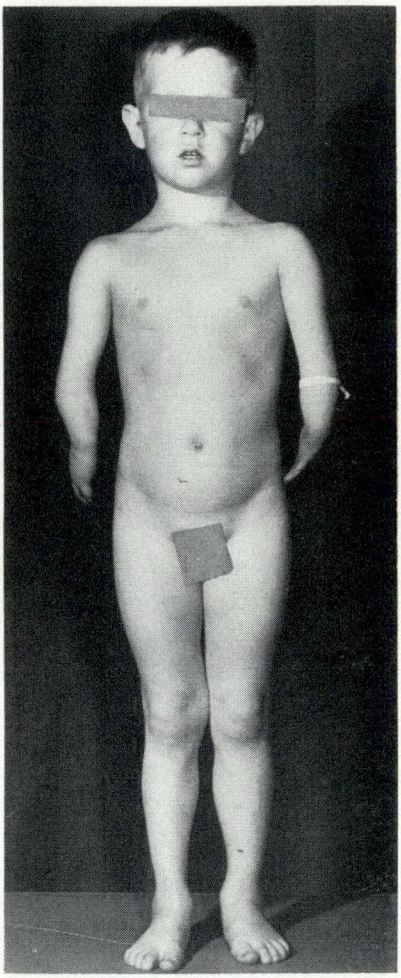

Fig. 3.—Same patient with bilateral absence of ulna at age six years. No other congenital anomalies (remarkable use of his upper extremities).

The hand defect varies considerably; it usually occurs on the ulnar side of the hand and may involve two, three or even four fingers. The presence of a thumb with a missing bone in the forearm, while suggestive of an absence of ulna, is not diagnostic. On rare occasions (Piulachs,[9] Mouchet and Pakouski[8] (1923)) an absent ulna may be associated with aplasia on the radial side of the hand. The fingers, however, can be difficult to identify and often exhibit syndactylism.

Distant congenital anomalies are rarely present, although congenital dislocation of the hip and bilateral absence of fibula have been reported.[1]

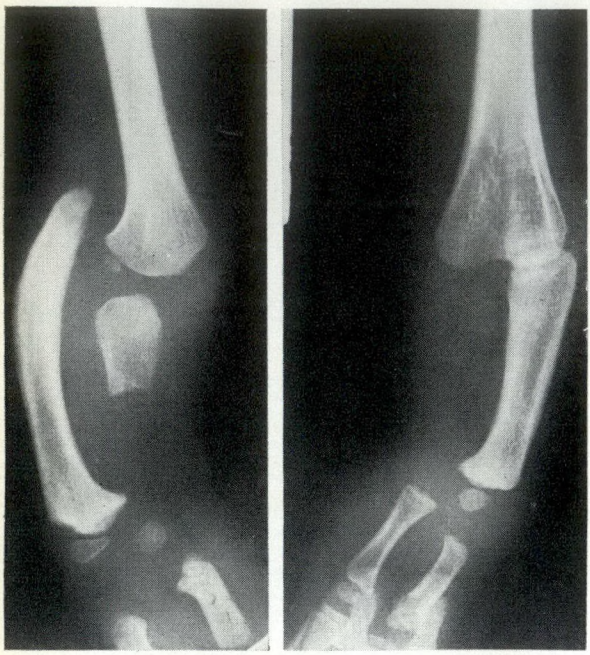

Fig. 4.—Radiographs at age six years now reveal bilateral *incomplete* absence of ulna; Type 2 on right, i.e., proximal dislocation of head of radius; Type 3 on left, i.e., radiohumeral synostosis.

Treatment

Treatment is symptomatic; certain cases will require correction of the hand anomaly, while others will be mostly disabled by their elbow.

Vitale[12] (1952) has recently applied Groves' principle of constructing a single radio-ulnar bone to this anomaly. The application of this method is limited to those few cases where elbow movement is satisfactory and where a proximal segment of ulna is present.

Fig. 5.—Pincer function of hand improved by correction of syndactylism and rotation osteotomy of radial metacarpal bone (left). Right side has since been similarly corrected.

Case Report

D.L. was the second sibling, after a normal pregnancy and delivery. There was no family history of congenital deformities. The anomalies present at birth were limited to the upper extremities (Fig. 1). Both upper limbs were deformed and shortened. There was mild limitation of movement at the right elbow joint, while the left side was fused in almost complete extension. The wrists showed good range of movement and almost no deformity. There was minimal manus vara on the left side. The hand deformities were comparable; three fingers were missing, apparently from the ulnar side. Syndactylism was present on both sides, more marked on the left. Radiological examination at the age of six months (Fig. 2) revealed complete bilateral absence of ulna with defective carpus and hand.

He was seen again at six years of age (Fig. 3). His clinical picture was essentially unchanged. His radiological appearance, however, revealed an interesting progression of events. The true picture of bilateral incomplete absence of ulna was now obvious. Radiohumeral fusion was complete on the left side (Type 2) while the radius was dislocated on the right side (Type 3). The lower epiphysis of the radius was deformed. The carpus was incomplete, but not dislocated. The unwary may at first confuse this radiological picture with the more common congenital absence of the radius.

Treatment of this patient so far has been limited to correction of his syndactylism and rotation osteotomy of the radial finger. This has improved the pincer action of his hands. His left elbow may be later improved by a flexion osteotomy of the humerus, or by an arthroplasty. The satisfactory function of his right arm does not warrant the construction of a single radio-ulnar bone.

Summary

The incidence, pathology and clinical picture of congenital absence of the ulna have been reviewed briefly. An addition has been made to the literature.

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Résumé

L'absence congénitale du cubitus vient après l'absence de l'humérus comme rareté; Rabaud et Havelocque, en 1924, en ont trouvé 71 cas dans la littérature. L'anomalie qui est souvent méconnue, est fréquemment unilatérale et affecte surtout le côté droit. Kummel en 1895 en a rapporté trois variétés: (1)Articulation radio-humérale normale avec radius normal ou légèrement incurvé. (2) Ankylose radio-humérale; avec fusion au radius ou à l'humérus du fragment proximal du cubitus s'il y en a un. (3) Dislocation proximale du radius.

Dans le membre normal, le cubitus sert surtout à la stabilité de l'articulation du coude, alors que le radius offre la surface articulaire la plus importante du poignet. Dans le cas d'absence congénitale du cubitus, l'articulation du coude est en général anormale.

Les lésions de la main varient considérablement et se retrouvent surtout du côté cubital, avec lésion de deux, trois ou quatre doigts. D'autres anomalies congénitales peuvent coexister, comme à la hanche ou aux péronés, mais elles sont excessivement rares.

Le traitement est symptomatique et s'appliquera à la main ou au coude, selon le cas. Vitale a préconisé en 1952 un traitement par fusion qui s'emploie lorsque l'articulation du coude est satisfaisante et lorsqu'il y a un moignon proximal du cubitus.

Les auteurs rapportent un cas d'absence congénitale des deux cubitus avec absence des trois doigts internes des deux côtés et syndactylisme. Le traitement a consisté en une correction du syndactylisme avec ostéotomie et rotation de l'index pour assurer la préhension. Le coude gauche sera traité plus tard. Les mouvements du coude droit sont suffisants et n'indiquent pas la fusion radio-cubitale.

TRAITEMENT CHIRURGICAL DE LA RECTO-COLITE ULCERO-HEMORRAGIQUE (Surgical Treatment of Ulcerative Colitis). E. Delannoy and M. Martinot, University of Lille, France. 120 pp. Illust. Masson et Cie, Paris, 1957. 1.400 Fr. fr.

Dans leur étude du traitement chirurgical de la recto-colite ulcéro-hémorragique, les auteurs, MM. E. Delannoy et M. Martinot, de la Faculté de Médecine de Lille, rapportent 26 cas de recto-colite ulcéro-hémorragique et ils en étudient les différentes formes de traitement. Après avoir classifié les formes chirurgicales de la recto-colite ulcéreuse, les auteurs discutent les diverses interventions proposées, depuis l'appendicostomie jusqu'à la colo-proctectomie, en passant par les interventions dites à visées pathogéniques, telle la vagotomie. Se basant sur les résultats obtenus, Delannoy et Martinot se prononcent en faveur de la colo-proctectomie avec iléostomie abdominale. On peut en conclure que cette dernière intervention semble être universellement reconnue comme étant la seule qui puisse guérir définitivement le patient.

La technique de Devine et Webb, pour la conservation totale de la fonction sphinctérienne anale, est signalée. Elle semble satisfaisante, mais le petit nombre des observations rapportées et un manque de recul empêchent d'en apprécier la valeur.

Les auteurs du *Traitement chirurgical de la recto-colite ulcéro-hémorragique* paraissent beaucoup moins favorables que nous le sommes au Canada envers la technique d'iléostomie de Crile et de Turnbull. Cette réserve faite, reconnaissons que, dans son ensemble, ce petit livre de 104 pages, bien illustré, est véritablement à jour. Toutefois, il n'apporte rien de neuf. Une bibliographie indique quelque 175 articles relatifs au traitement chirurgical de la recto-colite ulcéro-hémorragique. La plupart ne sont pas de sources françaises, mais étrangères, ce qui nous porte à croire que, depuis la fin de la guerre, l'école française a largement subi l'influence de la médecine d'outremer. C'est d'ailleurs ce que constatent MM. Delannoy et Martinot.

HERNIA THROUGH THE FORAMEN OF WINSLOW: REPORT OF A CASE, WITH REVIEW OF THE LITERATURE*

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Introduction

Hernia into the lesser peritoneal sac through the foramen of Winslow is so rare that observation of a single case has in the past been considered justification for reporting it, together with a review of the literature. This paper continues the tradition. In the case to be presented the diagnosis was not made preoperatively, although in retrospect one feels that if those in attendance had been familiar with the description in the literature reviewed below, a preoperative diagnosis might have been made. Therefore it seems profitable to present once more the description of a single case and to bring the review of current literature up to date (1957).

Literature

Hernia through the foramen of Winslow was first described in 1834 by Blandin.[2] Extensive reviews of the literature were carried out in 1924 by Ullman§ and in 1927 by Dewis and Miller.¶ These reviews have repeatedly been referred to by later authors, and have not been studied in the original for this paper. The most complete recent reviews are those of Silverstone[10] in 1939 and St. John[12] in 1954. The supplementary bibliography to our paper contains all the references discovered since 1939 which are not included in the bibliography to St. John's paper. Silverstone gave the total number of reported cases as 55, including his own. Dorian and Stein[6] (1954) quote Lavard and Chevret[9] (1949) to the effect that there were then less than 70 cases reported. This agrees with our search of the literature since 1939. Smoot[11] (1952) gave the figure as less than 80 and after a further search of the literature up to the end of 1957 the total is still not over 80, unless several of the reports not available to us report more than one new case.

Incidence

St. John states that the age incidence of reported cases is from 15 months to 77 years, with no sex preponderance. Some of the earlier authors say that this hernia is commoner in males than in females in the ratio of 2:1. These figures may be reconciled by the fact that strangulation of the contents of the hernia is said to be more common in males,[1] and until recently this hernia attracted attention only when strangulated. With increased interest, largely by radiologists,[3, 5, 12] the diagnosis has been made in a larger number of asymptomatic cases and others with mild symptoms short of strangulation.

Etiology

Moynihan and Dobson ("On retroperitoneal hernia", 1906) are quoted in most of the subsequent papers. They stated that one or more of the following abnormalities must be present in order to permit hernia through the foramen of Winslow:

1. Persistence of a common mesentery for the entire intestinal tract.

2. Absence of secondary fusion of the right colon mesentery to the posterior body wall.

3. Exceptionally large foramen of Winslow.

4. Unusually long mesentery of the small bowel.

To these Hollenberg[7] added another factor. He described the unusually long right lobe of liver which apparently served to direct the cæcum towards the foramen in his case. Precipitating factors may be anything which increases intra-abdominal pressure and also forward flexion of the trunk.[1]

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§Ullman, A.: Hernia through the foramen of Winslow, *Surg. Gynec. & Obst.*, **38:** 225, 1924.
¶Dewis, J. W. and Miller, R. H.: Hernia through foramen of Winslow, *Surg. Gynec. & Obst.*, **45:** 95, 1927.

Signs and Symptoms

The signs and symptoms are those of obstruction and strangulation, and are not characteristic of this special type of hernia. Some have noted, as we did, that abdominal distension may be limited to the upper abdomen. In a number of cases the pulse has been paradoxically slow, i.e. 60-70 and rarely over 80; but this is not so in all cases. It has been noted that vomiting is not a common symptom.

Diagnosis

Until recent years the diagnosis of this condition was not made until laparotomy. Hollenberg[7] (1945) claimed the first preoperative diagnosis, which was made by barium meal and barium enema, but he evidently overlooked Jackson's report in 1930.[8] Jackson demonstrated most of the small bowel within the lesser sac, in a patient who had suffered chronic dyspepsia for 15 years. After operation and partial closure of an unusually large foramen of Winslow, the patient's symptoms were completely relieved. Smoot[11] (1952) and Cimmino[5] (1953) reported a case, apparently the first, in which a preoperative radiographic diagnosis was made without administration of barium. The basis for the diagnosis was the finding of gas and fluid levels in the lesser sac where the fluid levels did not conform to the anatomical boundaries of the lesser sac.[4, 5] Dorian and Stein in 1954 observed a gas bubble to the right of the stomach in plain films but did not appreciate the significance at once. They were however familiar with the observations of Smoot and Cimmino and were able to make the diagnosis preoperatively.

In our case the only radiographic finding was the presence of a large gas bubble in the region of the stomach. This was reported as a gas-filled stomach but passage of a duodenal tube did not relieve the distension. The significance of this was not fully appreciated at the time, and operation was carried out before further investigation was considered. The presumptive diagnosis was volvulus of large bowel with displacement of the stomach. St. John discusses the subject of radiographic diagnosis at some length and gives the criteria for diagnosis without administration of barium.

Treatment

All cases with strangulation require operation. The manœuvres to be considered on confirming the diagnosis include:

1. Reduction of the hernia by traction on the bowel from outside the foramen.

2. Enterostomy to decompress the contents of the sac.

3. Dilatation of the foramen by gentle digital pressure. This technique, while always suggested, has not met with much success in the hands of those who have tried it.

4. Opening the lesser sac, either through the gastrocolic or gastrohepatic omentum. This procedure is almost always undertaken and may facilitate some of the other procedures already mentioned. For example, enterostomy may be more effective in bringing about decompression if carried out through an opening in the lesser sac and on a loop of distended bowel rather than on collapsed bowel outside of the sac. Occasionally, it may be possible to reduce the hernia without enterostomy and decompression by combining traction from without with compression of the mass of bowel from within the lesser sac.[1]

5. Mobilization of the duodenum would appear to be a logical way of enlarging the foramen when necessary, but, in the cases most requiring it, the approach to perform duodenal mobilization would probably be blocked by the bowel leading towards the foramen. As a matter of record, this procedure has not been successfully carried out in the living patient.

Of these measures the two most valuable appear to be enterostomy for decompression, and opening the lesser sac. This latter step should probably always be taken to confirm the diagnosis, because in one recorded case the contents of the lesser sac entered through a rent in the transverse mesocolon and a loop of small bowel again issued from the foramen of Winslow into the greater sac. Obviously such a case would not respond well to an attempt to reduce the contents into the general cavity

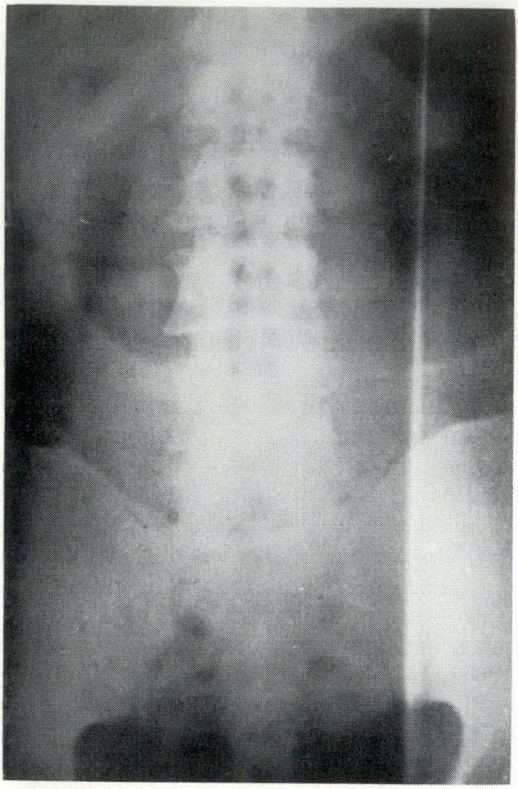

Fig. 1.—Plain film taken with the patient in the supine position.

by traction on the loop issuing from the foramen of Winslow.

The results of surgical treatment in a promptly diagnosed case should be as satisfactory as those in other types of intestinal obstruction or strangulation. The statistics available show much poorer results since most cases were not diagnosed early until recent years. Silverstone saw records for 44 patients, in whom only 33 operations were performed. Of these only 15 recovered. Silverstone's own patient was not operated on until five days after the onset of symptoms, and did not recover.

Report of a Case

W.B., a white man 63 years old, attended the emergency department of St. Joseph's Hospital at 3.30 p.m., July 7, 1956. His complaint at this time was abdominal pain of 60 hours' duration, and absolute constipation since the onset of abdominal pain.

Although pensioned and not doing any steady work, the patient was active physically and had enjoyed good health until the onset of his pain. Two days before coming to hospital he wakened in the morning and, according to his usual habit, had a bowel movement before preparing his breakfast. While eating breakfast he experienced a sudden severe lower abdominal pain. He returned to the bathroom and passed a very small liquid stool; after this there was no further passage of stool or flatus up to the time of admission to hospital. After this stool the pain subsided, but was not completely relieved. It remained fairly mild, but fluctuated in intensity for the next two days. He had little appetite but when he drank a cup of tea about 12.00 noon on the day of admission the pain became steady and severe. He described the pain as commencing in the midline just above the symphysis pubis and extending upwards to the epigastrium. He could not lie still but was not relieved by moving about.

Inquiry revealed no other symptoms of significance. The only abnormalities noted were some shortness of breath on considerable exertion and the presence of a right inguinal hernia since 1942. He was accustomed to wearing a truss when performing heavy work but did not find this necessary otherwise.

He was an alert robust man past middle age, grunting with pain, and appeared to be in considerable distress. Temperature 98° F., pulse 84, respirations 24, blood pressure 160/100 mm. Hg. His head and neck showed no significant abnormalities. Chest and cardiovascular system were normal on percussion and auscultation.

The abdomen was moderately obese and appeared distended, particularly in the epigastrium, where a distinct rounded, tense, tympanitic mass was present. There was tenderness over the entire abdomen. No localized tenderness, no other masses and no enlarged organs were detected. The inguinal rings were wide, with impulses present on coughing, but no hernia was actually demonstrable. Rectal examination revealed no bowel lesion. There was no blood or fæces on the examining finger. The prostate was firm and slightly enlarged.

Urinalysis: Reaction acid, S.G. 1.030, albumin 1+, sugar absent, microscopically 3-5 white cells per high power field and a few granular and hyaline casts. Hæmoglobin value 13.8 grams per 100 ml.

Plain films of the abdomen made in three positions showed a large gas bubble in the general region of the stomach (Fig. 1). This was the only obvious finding, and although search was made for gas and fluid levels in the remainder of the abdomen, none were

seen. The radiologists reported this large gas bubble to be in the stomach but passage of a Levine tube failed to relieve the distension. At this point the patient was prepared for operation and no further radiographic examinations were carried out. The preoperative diagnosis was "acute intestinal obstruction, possibly due to volvulus of large bowel".

Operative findings

On July 7 at 10.00 p.m. the abdomen was opened through an upper paramedian incision and the empty stomach was found pushed forward against the anterior abdominal wall by a large tympanitic mass filling most of the lesser sac. Because of the patient's obesity, it was impossible to identify the contents of the lesser sac until the gastrohepatic omentum was opened. It then became obvious that the distended bowel in the lesser sac included the cæcum and a considerable portion of the ascending colon together with about 10 inches (25 cm.) of the terminal ileum. This loop of bowel entered the lesser sac through the foramen of Winslow. The mesocolon was intact. The cæcum was distended to about 6 inches (15 cm.) in diameter. Enterostomy was performed and the colon decompressed. The enterostomy was then closed and the hernia was reduced without difficulty. With the cæcum restored to the right lower quadrant it did not seem feasible to perform any further manœuvre to prevent recurrence of the hernia. It was felt that there would be sufficient general adhesion formation to provide some fixation in the position in which the organs were left at the conclusion of laparotomy. A considerable portion of the very obese greater omentum was resected to facilitate closure of the abdominal wound.

The patient's postoperative course was quite stormy and he required Wangensteen suction and intravenous therapy for a number of days. Later in his postoperative course he developed an extensive wound infection which required removal of most of the skin sutures for drainage. Secondary suture of the skin was carried out on August 7, and he was finally discharged, after six and one-half weeks in hospital, on August 22. He was seen again in the outpatient department on August 31. He had no gastrointestinal symptoms but showed some weakness of his abdominal wall at the site of the incision. This was causing no symptoms and no further treatment was contemplated at that time.

Discussion

This case in retrospect appears fairly typical. There was a clear-cut history of bowel obstruction, and it was appreciated that with the rather atypical history and x-ray findings some one of the rarer causes of intestinal obstruction was probable. The radiographs did not present the typical appearance of gas-filled loops of bowel occupying the lesser sac, as described by Cimmino, but the fact that a Levine tube inserted in the stomach failed to decompress the patient's abdomen should have suggested to us that the gas was in fact behind the stomach. In this case there was an obvious intestinal obstruction, and laparotomy for relief of the obstruction was clearly indicated, so further attempts at radiographic diagnosis were not considered.

Summary

Hernia into the lesser sac through the foramen of Winslow is a rare form of internal hernia. Less than 80 cases have been reported in the world literature since 1834.

The literature has been reviewed, and for completeness a supplementary bibliography containing references to those papers unavailable to us is included.

Etiology, diagnosis, and treatment are discussed briefly, and one case is reported. This was the case of a 63 year old male who was successfully operated on after nearly three days of obstructive symptoms.

Diagnosis of this hernia is usually made at laparotomy for intestinal obstruction, but can sometimes be made preoperatively with the aid of suitable radiographic examination.

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Résumé

Les hernies à travers l'hiatus de Winslow sont très rares; la littérature ne compte qu'une vingtaine de publications sur le sujet. Il est à signaler que la première observation remonte à 1834, et que quelques auteurs réussirent à en faire le diagnostic avant l'intervention.

Ce syndrome se rencontre plus fréquemment chez les hommes que chez les femmes. Il semble que certaines malformations congénitales en soient les principaux facteurs étiologiques. Telles, la persistance d'un mésentère commun tout le long du tractus intestinal, l'absence de soudure du mésocolon droit à la paroi abdominale postérieure, une largeur excessive du hiatus de Winslow, un mésentère anormalement long ou enfin (facteur récemment invoqué par Hollenberg) un lobe hépatique droit de grandeur anormale qui aiderait le cæcum à glisser vers le hiatus de Winslow.

La symptomatologie est celle d'un iléus, sans caractéristiques. Le diagnostic différentiel est évidemment très difficile et n'est, souvent, possible qu'à l'opération. Le traitement est toujours chirurgical. Les manœuvres que l'opérateur devra envisager sont: (1) la réduction par simple traction de la hernie; (2) l'entérostomie en vue de décomprimer, si besoin est; (3) la dilatation des bords du hiatus, qui devra se faire prudemment du bout du doigt; (4) l'ouverture du sac en passant à travers le ligament gastro-colique ou le ligament gastro-hépatique. La mobilisation du duodénum en vue d'élargir l'orifice herniaire est ici trop complexe.

Le cas que présentent les auteurs est celui d'un homme de 63 ans qui souffrait, à l'admission, de douleurs intestinales et de constipation depuis 60 heures. La douleur siégeait sur la ligne médiane entre la symphyse pubienne et l'épigastre. Le patient n'avait aucun antécédent pathologique; il présentait une température à 98° F. et un pouls à 84. L'abdomen était très distendu et tympanique, surtout dans sa moitié supérieure. Des radiographies à vide, prises dans trois positions montrèrent une volumineuse ombre gazeuse dans la région gastrique mais rien d'autre. On soupçonna un volvulus du gros intestin et l'intervention d'urgence fut décidée.

Une laparotomie paramédiane permit de trouver l'estomac vide et repoussé contre la paroi antérieure par une énorme masse intestinale. Après avoir ouvert le ligament gastro-hépatique, on put reconnaître que ce paquet, formé du cæcum, du colon ascendant et d'une partie du grêle, était étranglé dans l'hiatus de Winslow. On pratiqua une entérostomie de décompression, ce qui permit une réduction relativement aisée. On ne fit aucune manœuvre spéciale en vue de fixer les anses intestinales en place, comptant sur la création des adhérences post-opératoires habituelles.

Les suites furent assez compliquées: une aspiration continue due être installée et maintenue pendant plusieurs jours; de plus la plaie abdominale s'infecta et il fallut enlever la plupart des points de suture. Une deuxième suture dut être exécutée un peu plus tard. Le malade finit par se rétablir cependant, et jouit maintenant (trois ans après) d'une bonne santé.

MALAKOPLAKIA: REPORT OF A CASE*

N. C. CARRUTHERS, M.D., *Toronto*

THE TERM MALAKOPLAKIA was introduced by von Hanseman in 1903. The disease was first described by Michaelis and Gutman in 1902. To date, 69 cases have appeared in the literature.

CASE REPORT

Mr. W.W., aged 62, was first admitted to Sunnybrook Hospital on March 25, 1958, with complaints of backache, urinary frequency, dysuria, and terminal hæmaturia of five years' duration. He had a history of latent syphilis in 1940, perforated peptic ulcer in 1943, and recurrence of ulcer symptoms in 1951.

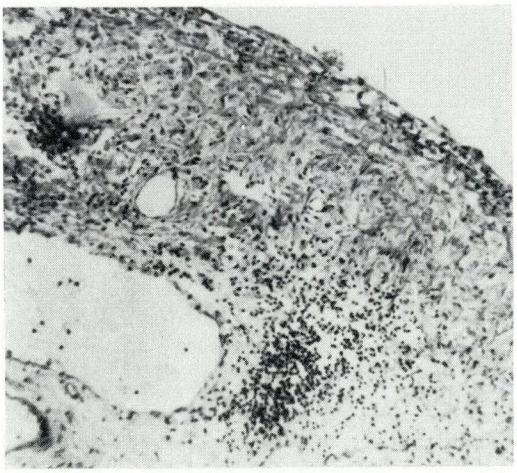

Fig. 1.—Lower power microscopic view through a plaque showing associated cystitis.

Physical examination was essentially negative. Digital examination of the prostate revealed slight, benign enlargement. Laboratory findings were normal except for the growth of *E. coli* in the urine culture. Intravenous pyelography showed a smooth deformity of the left middle calyx considered to be due to a cyst.

At cystoscopy, several raised plaque-like areas were seen within the bladder and at the vesical neck; the prostate was enlarged. On April 1, 1958, transurethral resections of the prostate and the plaques were carried out.

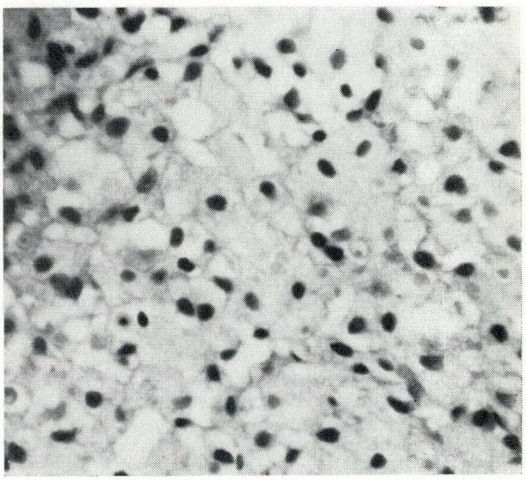

Fig. 2.—High power microscopic view showing large macrophages.

The pathologist reported: "Sections of tissue from urinary bladder show a granulomatous inflammatory reaction with masses of closely packed, large, oval and polygonal, eosinophilic macrophages filling and widening the tunica propria. The large cells have a granular cytoplasm and many contain basophilic and some eosinophilic double-ringed structures which superficially resemble yeast forms of fungi. These are the so-called calcospherites as seen in malakoplakia. Only fragments of mucosa are present on the surface. Clusters of lymphocytes, a few plasma cells, and some

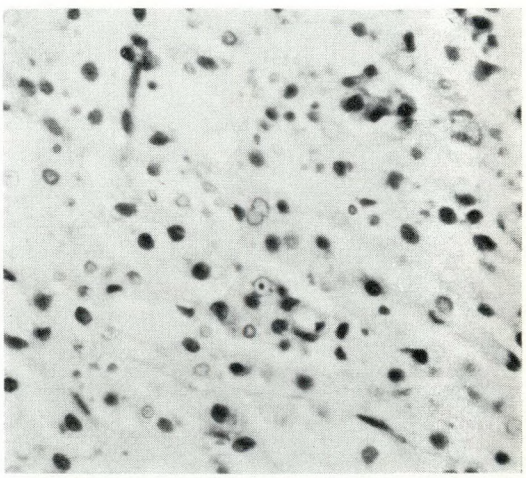

Fig. 3.—High power microscopic view showing several calcospherites or Michaelis-Gutman bodies.

*From the Department of Urology, Sunnybrook Hospital, Toronto.

hæmosiderin pigment are present in the base of the tunica propria". The pathological diagnosis was: "Malakoplakia of urinary bladder, bladder neck, and prostatic ducts" (Figs. 1, 2, and 3).

The patient was discharged on April 14, 1958, after an uneventful postoperative course and was readmitted on July 29, 1958, for follow-up examination. There were no urinary complaints but cystoscopy showed two new plaques on the bladder floor, and the urine cultured a heavy growth of *E. coli*. Sulfonamide therapy was given and arrangements were made for fulguration of the plaques after a three-month interval.

Discussion

The lesions of malakoplakia may affect any part of the urinary tract including renal and prostatic parenchyma and may even be found in the extra-urinary tissues as reported by Scott and Scott.[6] Females are more commonly affected than males in the ratio of 4:1. The highest incidence of cases is in the 40 to 49 year group.

The etiology of malakoplakia is obscure. The patients usually have chronic disease and urinary tract infection. A tuberculous etiology has been suggested and King[5] reports success with anti-tuberculosis therapy in one case.

Pathologically, the lesions are benign granulomata consisting of discrete pinkish, raised, and usually small plaques. Microscopically, the plaques are characterized by collections of histiocytes (von Hanseman cells) containing Michaelis-Gutman bodies or calcospherites. These may be mistaken for *Cryptococcus neoformans* but can be differentiated by demonstration of calcium and iron, using trichrome methylene blue and von Kossa stains.

Treatment has been by fulguration and has been generally successful. Urinary infection should be cleared, if possible. One fatal case was reported by Scott and Scott.

Summary

An additional case of malakoplakia involving bladder, bladder neck, and prostate is presented. Lesions recurred after a three month interval. Attention is drawn to this disease as a cause of hæmaturia to be distinguished from malignancy in the urinary tract.

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Résumé

On rapporte le cas d'un homme de 63 ans, qui souffrait de douleurs lombaires, de dysurie et d'hématurie. L'examen général ne dénote rien, la prostate est très légèrement hypertrophiée. Les épreuves de laboratoire sont normales sauf la culture d'urine qui montre la présence de colibacille. A la pyélographie intraveineuse, on trouve une légère déformation du calice moyen gauche. La cystoscopie permet de voir plusieurs plages surélevées sur le col vésical, la prostate est hypertrophiée. Des résections transuréthrales de la prostate et de ces plages sont faites. L'examen histologique conclut à des lésions malacoplasiques de la vessie, du col vésical et des canaux prostatiques. Les suites opératoires furent bonnes mais de nouvelles plaques apparurent trois mois plus tard. L'étiologie de cette affection est obscure. Les femmes sont plus touchées que les hommes, dans un rapport de 4 à 1. Microscopiquement, les plaques sont constituées par des amas histiocytaires. Le traitement est l'étincelage de toutes les lésions, si cela est possible.

POSITIVE PRESSURE REDUCTION OF FRACTURES OF THE STERNUM

LUKE TESKEY, JR., M.D.,* *Toronto*

SINCE THE PUBLICATION by Avery, Morch and Benson of their method of reduction of the stove-in chest by "hyperventilation", the author has wondered whether some sternal fractures could be reduced by producing one incident of increased intrathoracic pressure. Transverse fractures of the sternum seem to meet the requirements. Two cases are presented which illustrate that these transverse fractures of the sternum may be reduced by one incident of increased intrathoracic pressure, and that such reduction of these fractures is stable and maintained without fixation.

CASE 1 (Fig. 1).—Mr. A.P., aged 73, was admitted to the Northwestern General Hospital, Toronto, on November 7, 1957, after a car accident. Together with his fracture dislocation of the sternum, he suffered fractures of the 2nd, 3rd and 4th left ribs and a fractured left patella. On the day of admission the fracture of the sternum was reduced by induction of general anæsthesia, positive pressure being applied through the intubation tube by pressure on the gas bag. The reduction was stable and the mechanics of the chest greatly improved. No fixation was used. Unfortunately no post-reduction films were obtained. The following day, because of increasing mucus, a bronchoscopic aspiration was carried out. The next day, November 9, the patient's respiration was still laboured and a tracheotomy and chest wiring were performed to stabilize the stove-in chest. The clinical picture improved until November 11, when he developed a bout of coughing followed by shock and death in spite of multiple transfusions. Post-mortem examination revealed a rupture of the arch of the aorta, which was arteriosclerotic.

CASE 2 (Fig. 2).—Mr. G.M. was admitted after a blow on the chest from a piece of lumber thrown from a power saw. This man was in such acute distress that he could not lie down, and was almost violent because of a mild anoxia. The deformity was palpated and a radiograph obtained.

On seeing this radiograph (Fig. 2) the author resolved to try to increase the intra-

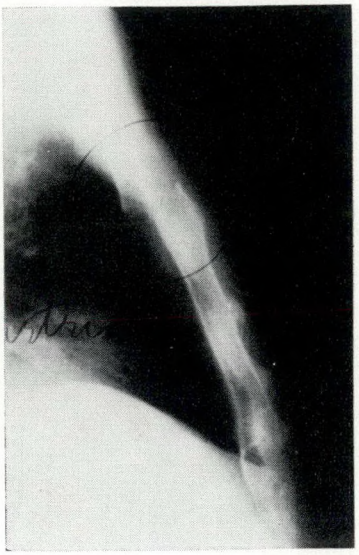

Fig. 1.—Case 1. Radiograph showing fracture dislocation of the sternum on admission to hospital.

thoracic pressure without the aid of machinery. The manœuvre of forced total inspiration followed by forced expiration against the closed mouth and nose came to mind, and was tried.

The fracture was infiltrated with 2 c.c. of cyclaine. In seconds, the pain was relieved and

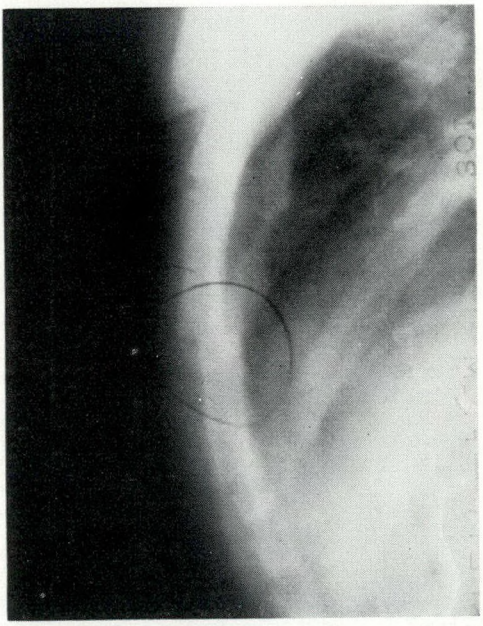

Fig. 2.—Case 2. Radiograph taken on admission to hospital.

*Surgical Staff, Northwestern General Hospital, Toronto.

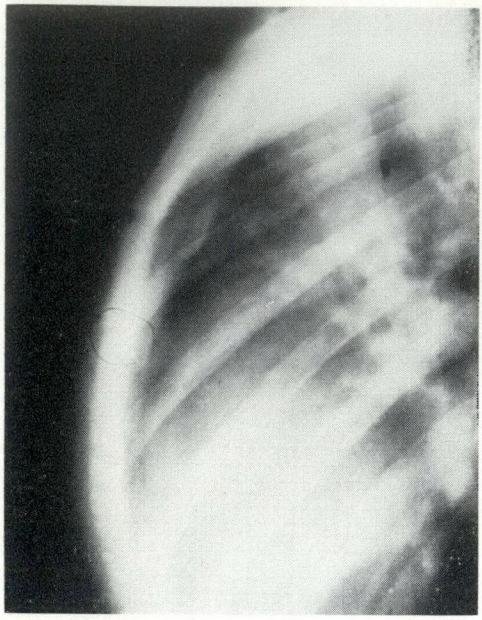

Fig. 3.—Case 2. Post-reduction radiograph.

the patient became co-operative. He was instructed to take as big a breath as he could, then to close his mouth, to pinch his nose and attempt to breathe out. The fracture was reduced and the deformity was no longer palpable. The post-reduction radiograph is shown (Fig. 3). No fixation was required, the position did not deteriorate and the patient resumed work in about one month.

The principle of positive pressure is new to treatment of chest injuries. It is very effective and can be produced: (a) by maximum inspiration followed by forced expiration against the resistance of the closed mouth and pinched nose (the Valsalva manœuvre); (b) by the anæsthetic machine after intubation.

It is hoped that the simple method of local infiltration followed by the simple manœuvre outlined may become a useful method of treating transverse fractures of the sternum.

Summary

A new method of reducing fractures of the sternum is presented. This method should be tried before open operations are embarked upon.

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Toronto.

Résumé

Deux cas sont présentés dans lesquels une fracture transversale du sternum a pu être réduite par une simple augmentation de la pression intra-thoracique.

Le premier cas est celui d'un homme de 73 ans hospitalisé après un accident d'automobile pour des fractures de côtes et du sternum. Cette dernière fut réduite sous anesthésie générale avec intubation par création d'une pression positive dans l'appareil. La réduction était stable et aucune fixation ne fut nécessaire.

Dans le second cas il s'agit d'un patient qui avait reçu un bloc de bois sur le thorax. Il présentait une fracture sternale avec grosse déformation. On procéda à une anesthésie locale du foyer et l'on fit inspirer le malade aussi profondément que possible: puis, bouche et narines fermées, on lui fit faire des efforts d'expiration. Cette manœuvre suffit à assurer une réduction stable.

Cette méthode simple est efficace: il est intéressant de la connaître car elle peut être de la plus grande utilité dans les traumatismes du thorax.

OPERABLE HERZLEIDEN. Einführung in Klinik, Diagnostik und Operationsmöglichkeiten (Operable Heart Conditions. An Introduction to Clinical Features, Diagnosis and Scope of Operation). J. Jacobi and M. Loeweneck, Hamburg. 175 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1958. $11.40.

With the rise of cardiac surgery, it has become increasingly important for the general practitioner to recognize which cases should be referred to a surgeon. This will of course constitute only a small proportion of the cases in his care, and after operation they will revert to his care. The present work by Jacobi and Loeweneck of Hamburg is designed purely for the general physician. It is intended to teach him the scope of modern cardiac surgery, and to give him indications of the selection of suitable patients and the diagnostic aids which need to be employed. Hence, details of operations are briefly described, while differential diagnosis and clinical features of the various conditions are mentioned in some detail.

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BOOK REVIEWS

(See also pages 137, 199, 207 and 216)

OPERATIVE SURGERY. Volume 7. Breast and Genito-Urinary System. Edited by Charles Rob and Rodney Smith. 273 pp. Illust. Butterworth & Co. (Canada) Limited, Toronto, 1958. $21.50.

The arrival of Volume 7 of this already well-received series marks the last but one of this reference work in operative surgery. It is devoted to breast and genito-urinary surgery.

The section dealing with the former will have wide appeal amongst general surgeons; it includes minor as well as major procedures. The account of radical mastectomy is particularly good and should be read by all general surgeons. It shows in detail what is expected in this all too common dissection. There is wide diversity of opinions and practice in regard to this operation; one probable factor is the variety of results obtained by different operators. This analysis serves to remind all surgeons of the completeness and radical nature of the operation.

A long section deals with the less common mammaplasty and might be better included under the section dealing with plastic surgery.

Genito-urinary surgery is covered in the remaining 275 pages and constitutes a veritable textbook in itself. An innovation is a section dealing with surgery of access to the kidney as distinct from renal surgery itself. Routes and their uses are clearly demonstrated.

The remainder of this chapter deals with all the common procedures on each area of interest to the urologist. The drawings are done with the same clarity as in previous volumes—the text is concise but adequate and is well co-ordinated with the illustrations, so that it is not necessary to turn to other pages to follow the contents quickly. This volume only serves to emphasize once more the value and completeness of the series; the reviewer has no hesitation in recommending it to all surgeons.

OPERATIVE SURGERY. Volume 8. Neurosurgery, Eyes, Ear, Nose and Throat. Edited by Charles Rob and Rodney Smith, London, England. 196 pp. Illust. Butterworth & Co. (Canada) Limited, Toronto, 1958. $19.50.

The appearance of Volume 8 completes this monumental work on operative surgery, written throughout with a view to serving the needs of the general surgeon rather than the specialist in any one field. It is therefore understandable that the section on neurosurgery should be abbreviated to a few emergency procedures such as depressed fractures of skull, penetrating brain injuries and extradural and intradural hæmorrhage. The inclusion of trigeminal sensory-root injection is a little more difficult to appreciate.

The second section, dealing with ophthalmology, includes many procedures which certainly do not fall within the sphere of interest of general surgeons. The surgery of retinal detachment, surgery of the ocular muscles, and cataract surgery are the province of specialists in ophthalmology in this country.

Part Three deals with ear, nose and throat problems. What has been said before applies here too, in that few of the problems discussed will interest the general surgeon and very few are emergency operations. This section, however, will have a great appeal to the specialist in this field and perhaps as a reference work for students at the resident level.

The same concise format is followed throughout as in the previous seven volumes. The illustrations are helpful and accurate and point up the essential features of the subjects. The style is entirely delightful and this volume should be of special interest to workers in these particular fields.

BASES PHYSIO-BIOLOGIQUES ET PRINCIPES GENERAUX DE REANIMATION (Physio-Biological Bases and General Principles of Reanimation). H. Laborit. 273 pp. Illust. Masson & Cie., Paris, 1958.

Dans ce traité, l'auteur expose les principes généraux gouvernant la régulation des processus vitaux, de la cellule à l'organisme entier, régulation basée sur l'équilibre physico-chimique entre le milieu intérieur et le milieu intra-cellulaire. Toute agression est capable de perturber cet équilibre et d'engendrer une série de réactions dont l'ensemble revêt une forme unique quelles que soient les lésions spécifiquement en cause. L'étude de ces perturbations réactionnelles et les mesures thérapeutiques visant à leur correction font l'objet de l'aggressologie et de la réanimation.

La cellule, siège de phénomènes métaboliques, est le point de départ des changements réactionnels. Conséquemment les corrections nécessaires devront s'adresser aussi bien à la cellule qu'au milieu intérieur qui lui sert d'intermédiaire avec le milieu extérieur.

La formation d'ions $H+$ à partir des processus métaboliques intracellulaires, leur mobilisation par le système cardio-vasculaire et leur élimination par voies pulmonaire et rénale constituent l'idée dominante de l'ouvrage. Revue étant faite des phénomènes oxydo-réductifs cellulaires sous leur aspect bioélectrogénétique et biochimique, l'auteur analyse leur régulation en faisant ressortir l'importance du rejet constant du sodium et de la réintégration cellulaire du potassium. La réanimation devra donc assurer la protection des systèmes dont dépendent la formation et l'élimination de l'ion $H+$: protection des processus métaboliques par apport de substrats oxydables, protection du système respiratoire par respiration artificielle, expectoration artificielle, trachéotomie, etc., protection de l'émonctoire rénal. Quant au système cardio-vasculaire, la protection en sera assurée par l'inhibition de la réaction sympa-

(Continued on page 220)

(Continued from page 218)

thique et endocrinienne, par la réduction du sodium extracellulaire et la restauration de la masse sanguine. Cette protection peut être aidée par les méthodes d'inhibition réactionnelle et la réduction des processus métaboliques: une large place est donc réservée à l'hibernothérapie. Les problèmes de la sénescence et de la douleur sont abordés à la lumière du concept physiobiologique de la réaction à l'agression.

Cette tentative d'intégration de toute la physiopathologie dans ce concept unitaire de la maladie augure peut-être une discipline médicale nouvelle que d'autres recherches expérimentales et cliniques devront encore éprouver.

Ce volume comporte une bibliographie internationale très vaste et sa forme concise et claire en rendent la lecture attrayante.

TO WORK IN THE VINEYARD OF SURGERY. The Reminiscences of J. Collins Warren (1842-1927). Edited by Edward D. Churchill, Harvard University, Boston, Mass. 288 pp. Harvard University Press, Cambridge, Mass.; S. J. Reginald Saunders and Company, Limited, Toronto, 1958. $7.95.

This book is subtitled "The Reminiscences of J. Collins Warren (1842-1927)", but it is very much more than the memories of a great member of a great family of surgeons of Boston. It is the story of the three stages in the development of surgery: before anæsthesia, before antisepsis, and since. J. C. Warren's father performed the historic operation under ether at the Massachusetts General Hospital on October 16, 1846, when the author was four years old. He knew Virchow and Billroth and Lister, Paget and Thompson.

There are anecdotes of how money was obtained from John D. Rockefeller, J. P. Morgan and Mrs. Huntington to build Harvard Medical School. He tells of his colleagues Oliver Wendell Holmes, Bowditch, President Elliot, and Fitz who added the word "appendicitis" to medical nomenclature. In the modest sketches of the events he witnessed, the men he met and the dreams he made come true, there is the essence of history.

Dr. Churchill, who has added immeasurably to the interest of the original memoirs with footnotes and appendices, is the present occupant of two of J.C.W.'s appointments: professor of surgery at Harvard and chief surgeon at the Massachusetts General Hospital.

"To Work in the Vineyard of Surgery" is just about the whole story of surgery, told as only an eyewitness of those great decades could tell it.

THE SURGEON'S TALE. R. G. Richardson. 256 pp. Illust. George Allen & Unwin Ltd., London, 1958. 25s.

This book on the evolution of modern surgery was first planned by the publisher for the reader with no medical background. But the author protested that so technical a subject demanded a little more than a basic knowledge of medicine for adequate comprehension. A compromise has resulted in a nice blend of story-telling and teaching, with a glossary of terms and extensive index of names and subject matter. Dr. Richardson has achieved a book of interest to readers both within and without, but perhaps of most value to those on the fringe of the surgical profession.

The past 100 years of surgery, from the discovery of anæsthesia and antisepsis to open heart surgery and the latest attacks on cancer, have been traced. The book abounds in stories: stories of men whose contributions built surgery, stories of patients made famous by being the first to undergo new treatments, stories behind the discoveries that led to the biggest advances. Operative techniques are described, dates are given, and a wealth of fact has been gleaned from all the literature. For, as the author says, "Without the past the present is largely meaningless and the future cannot be divined."

MILESTONES IN MODERN SURGERY. Edited by Alfred Hurwitz, Professor of Surgery, State University of New York College of Medicine, New York, and George A. Degenshein, Assistant Attending Surgeon, Maimonides Hospital, New York. 520 pp. Illust. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1958. $15.00.

Most of this symposium consists of reprints of important papers which have appeared in the surgical literature, together with a commentary on their authors. Eleven surgical themes ranging from hæmostasis and anæsthesia to cardiovascular surgery are involved, and in each case a few key papers are reproduced in part or in whole.

The selection ranges from Ambroise Paré and Spencer Wells on hæmostasis to Matas, Carrel, Souttar and Gross on cardiovascular surgery. Fleming, Kocher, Halsted, Bassini, Billroth, Wangensteen and others are represented, and the book closes with an essay by Dunphy on changing concepts in cancer surgery, and two recent papers on the extracorporeal circulation and transplantation of kidney. The whole book constitutes a good survey of important topics in the development of modern surgery.

LEHRBUCH DER CHIRURGIE (Textbook of Surgery). Edited by H. Hellner and others. 1112 pp. Illust. 2nd ed. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1958. $20.00.

In 1957, the first edition appeared of a text on surgery with contributors from many German and Swiss university centres. Within 18 months of this first edition, a second one has been found necessary; this suggests two things, that the text has been very well received in German-speaking quarters and that the team

headed by Hellner, Nissen and Vossschulte is determined to keep the work up to date. In their preface the authors point to the fact that anatomical boundaries of surgery have now been reached, and advances will be dependent on findings in applied physiology. They have therefore laid stress on applied physiology and functional pathology in their text, with exclusion of anatomical detail and details of surgical techniques. The result is a readable and up-to-date text. That the world literature has been carefully studied is indicated in particular by the chapter on cardiac surgery by Koncz, in which Canadian and American work receive due consideration. Any German-speaking physician looking for a one-volume review of the whole of surgery should find his requirements met by this beautifully produced and well illustrated German text.

ALLGEMEINE UND SPEZIELLE CHIRURGISCHE OPERATIONSLEHRE (Textbook of General and Special Operative Surgery. Volume I, Parts I and II). Edited by Gerd Hegermann, Erlangen. 420 and 747 pp. Illust. 2nd ed. Springer-Verlag, Berlin, W. Germany, 1958. DM. 496 (Parts I and II).

A ten-volume set on surgery was published 32 years ago, after the First World War, by Professors Kirschner and Nordmann. At that time it caused a great sensation because it was the first such large publication in the general medical and surgical field. Today, three decades later, the first and second volumes of Dr. Kirschner's surgical work have been re-edited by Dr. Gerd Hegermann, professor of surgery at Erlangen and issued in a considerably revised form.

During these decades every branch of surgery, as well as its fundamental tenets, have altered. Not only concepts of narcosis, antisepsis, and surgical techniques, which appeared modern in Dr. Kirschner's first edition and were based on the experiences of the First World War, have changed significantly, but also their fundamentals. Altered as well are normal pathological anatomy, physiology, and physiological chemistry. Hegermann has re-edited the first two volumes of this work on a new basis. The two volumes deal with general surgery. The first deals with the most recent developments in plastic surgery, neurosurgery, bone surgery, and other special branches of surgery. The second volume discusses general and local anæsthesia, burns, hæmorrhage, transfusions, surgical infections, shock, thrombosis, and the dangers of surgical interference. Both volumes are augmented by very attractive, intelligible new illustrations, whose execution far surpasses that of the first edition.

Hegermann cites in the foreword the principle which Kirschner had expressed in the first edition, namely that the surgeon ought to operate not on what is technically operable but on what may and must be operated upon.

TUMOR SURGERY OF THE HEAD AND NECK. Robert S. Pollack, Clinical Instructor in Surgery, Stanford University School of Medicine, San Francisco, California. 101 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1957. $5.00.

In this concise handbook, the author takes for granted that his readers have achieved basic surgical knowledge and are competent to perform a standard radical neck dissection. He then describes briefly but accurately most of the other routine surgical procedures utilized in the management of oral cancer, carcinoma of the larynx, salivary gland lesions, and thyroid carcinoma. The operations are clearly illustrated by line drawings. The indications for surgical treatment and/or irradiation are discussed in a very sensible fashion, indicating that the author takes a reasonable and humane position in regard to the respective roles of these types of treatment.

Although this book does not claim to present anything new or original, it certainly succeeds in giving an accurate summary of a small but important branch of surgical care.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Atlas of Technics in Surgery. John L. Madden, New York Medical College, with 62 contributing authors. 648 pp. Illust. Appleton-Century-Crofts, Inc., New York, 1958.

Leistungen und Ergebnisse der Neuzeitlichen Chirurgie. Emil K. Frey zum 70. Geburtstag (Achievements and Results in Modern Surgery. Dedicated to Emil K. Frey on his 70th Birthday). 339 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1958. $20.00.

Management of Abdominal Operations. Edited by Rodney Maingot. 2nd ed. Vol. II. 1326 pp. Illust. The Macmillan Company, New York; Brett Macmillan Ltd., Toronto, 1957. $32.00.

The Management of Emergencies in Thoracic Surgery. John Borrie, University of Otego Medical School, New Zealand. 340 pp. Illust. Appleton-Century-Crofts, Inc., New York, 1958.

Notes de Techniques Chirurgicales de la Presse Médicale (Notes on Surgical Techniques from La Presse Médicale. Collected by Lucien Leger. 318 pp. Illust. Masson et Cie, Paris, 1958. 3.800 Fr. fr.

Treatment of Breast Tumors. Robert S. Pollack, Stanford University College of Medicine, San Francisco, and others. 147 pp. Illust. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1958. $6.00.

(Continued on page 222)

(Continued from page 221)

Head Injuries: Mechanisms, Diagnosis and Management. E. S. Gurdjian and J. E. Webster, Wayne State University College of Medicine, Detroit. 482 pp. Illust. Little, Brown and Company, Boston and Toronto, 1958. $14.00.

Crusader Undaunted. Dr. J. G. Geiger, Private Physician to the Public. Max S. Marshall, University of California School of Medicine, San Francisco. 246 pp. The Macmillan Company, New York; Brett-Macmillan Ltd., Toronto, 1958. $3.50.

Treatment of Cancer and Allied Diseases. Volume 1: Principles of Treatment. Edited by George T. Pack, Cornell University Medical College, and Irving M. Ariel, New York Medical College, New York. 646 pp. Illust. 2nd ed. Paul B. Hoeber, Inc., Medical Book Department of Harper & Brothers, New York, 1958. $22.50.

Rehabilitation of the Hand. C. B. Wynn Parry, Specialist in Physical Medicine, Royal Air Force, assisted by N. R. Smythe and L. E. Baker, with contributions from D. A. Brewerton and D. Brooks. 273 pp. Illust. Butterworth & Co. (Canada) Limited, Toronto, 1958. $9.00.

Cold Injury. Transactions of the Fifth Conference, March 10-15, 1957. Edited by M. Irené Ferrer, Columbia University College of Physicians and Surgeons, New York. 341 pp. Illust. Josiah Macy, Jr. Foundation, New York, 1958. $5.95.

Lesions of the Lower Bowel. Raymond J. Jackman, Mayo Foundation, Graduate School, University of Minnesota, Rochester, Minn. 347 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1958.

Electrolyte Changes in Surgery. Kathleen E. Roberts, Stanford University College of Medicine, San Francisco, Parker Vanamee, and J. William Poppell, Cornell University College of Medicine, New York. 113 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1958.

Allgemeine und Spezielle Chirurgische Operationslehre: Zweite Auflage, Erste Band, Teil II. (Textbook of General and Special Operative Surgery: Second Edition, First Volume, Part II). Gerd Hegermann. Edited by N. Guleke and R. Zenker. 747 pp. Illust. Springer-Verlag, Berlin, Goettingen, Heidelberg, W. Germany. 1958. DM 496.— for Part I and II.

Fortschritte der Kiefer- und Gesichts-Chirurgie. Ein Jahrbuch. Band IV. (Advances in Facio-Maxillary Surgery. A Year Book. Vol. IV.) Karl Schuchardt, Hamburg University. 441 pp. Illust. Georg Thieme Verlag, Stuttgart; Intercontinental Medical Book Corporation, New York, 1958. $29.55.

Operative Surgery. Vol. 8. Neurosurgery, Eyes, Ear, Nose and Throat. Edited by C. Rob, St. Mary's Hospital, London, England and R. Smith, St. George's Hospital, London, England. 196 pp. Illust. Butterworth & Co. (Canada) Limited, Toronto, 1958. $21.50.

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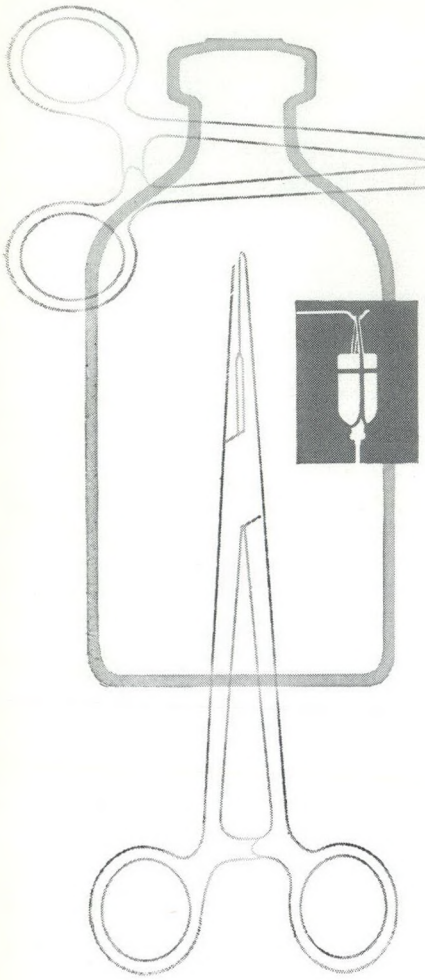

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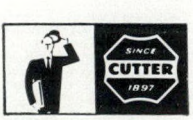

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